The Impact of a Mother's Wellbeing During Pregnancy on the Human Capital Endowment and Long Term Economic Outcomes of the In Utero Child

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Dissertation submitted in partial fulfillment of the requirements for the degree of
Doctor of Philosophy in the Department of Economics
in the Graduate School of Duke University
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Abstract

The goal of this dissertation is to provide new insights into how the social, physical and health environment during gestation affect the early and later life outcomes of the in utero child. This work is grounded in a biologically-informed model of in utero development and applies state-of the-art econometric methods to population-representative data in order to rigorously examine the impact of a mothers mental and physical wellbeing during the fetal period on both the early-life health and long-term economic outcomes of the in utero child. After a brief introduction, the second chapter reexamines the pioneering work by Douglas Almond (2006), which is thought to establish that in utero exposure to an adverse disease environment has a large, negative impact on health and socioeconomic prosperity that reaches well into adulthood. The analysis in this section casts doubt on the identification strategy used in that seminal work, and suggests that conclusions about the deleterious impact of in utero exposure to the influenza pandemic on socioeconomic prosperity in adulthood are, at best, premature. The third and fourth chapters delve into the topic of the impact of a mother's mental health during pregnancy on the birth outcomes of the in utero child. Utilizing two traumatic and unanticipated events, the terrorist attacks of September 11th, 2001 and the surge in Mexican Drug War violence, these chapters provide strong evidence that exposure to increased maternal anxiety has a significant negative impact on the early-life health of the in utero child.

To my wife. You are my strength and motivation. You are my life preserver, guiding light, and shelter from the storm.

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1

Introduction

Spurred by the pivotal work of David Barker, the fetal-origins hypothesis, which suggests the health of a mother during pregnancy is predictive of the long run health outcomes of the in utero child, has become a popular area of study in social sciences. Examining this relationship, though, is fraught with challenges related to the endogenous relationship between a mother's health and other unobserved characteristics correlated with fetal health. The second chapter of this dissertation, On the Long Term Effects of the 1918 U.S. Influenza Pandemic, is a reexamination of the seminal work by Douglas Almond on the impact of the U.S. influenza pandemic on the long-term wellbeing of children in utero during this worsened disease environment. The analysis that my co-author, Duncan Thomas, and I conducted suggests that the findings generated by this influential work are driven by the compositional change in parental characteristics of the "treatment" cohort brought on by several population processes occurring during the time period under study. Once these factors are controlled there appears to be no relationship between a mother's health during a child's gestation and the child's subsequent long term outcomes.

Taking the lesson regarding the delicacy and thoughtfulness that must be applied

when employing a natural experiment strategy to studying early-life health, the third chapter of this dissertation is an investigation of the impact of acute maternal anxiety exposure, brought on by the September 11th, 2001 terrorist attacks in the United States, on birth outcomes of in-utero children. When examining this question, it is imperative to avoid two identification pitfalls common in natural experiment studies of this topic: non-stress related negative externalities and post-event endogenous fertility selection. The results of this analysis suggest that the children exposed to the event while in utero were born significantly smaller and earlier than previous cohorts. Specifically, I find there is a relatively small shift in the mean birth weight (8-15 grams) of the exposed cohorts, but a sizable increase (5-10%) in the proportion of children born preterm and low birth weight amongst the exposed. Additionally, the timing of the effect provides evidence that intrauterine growth is specifically restricted by first trimester exposure to stress, while gestational age is most reduced by increased maternal psychological distress in mid pregnancy.

Having established a statistically significant link between birth outcomes and maternal anxiety brought on by an event that did not lead to direct victimization amongst the majority of the sample, this research moves to an ongoing conflict that regularly spills over into the lives of non-combatants; the Mexican Drug War. The fourth section of this dissertation, The Mexican Drug War and Early-Life Health: The Impact of Violent Crime on Birth Outcomes, utilizes the rich longitudinal data of the Mexican Family Life Survey in order to estimate the effect of the recent, rapid, and unprecedented rise in violence in Mexico on the early-life health of children born to mothers exposed to conflict while pregnant. Employing a maternal fixed effect, intent-to-treat approach, I estimate that a mother's experience of local violence early in gestation leads to statistically and economically significant adverse effects on the birth outcome of the in utero child. The estimates, across multiple samples and specifications, consistently indicate that exposure, early in gestation, to the average

increase in local violent crime in Mexico between the pre-escalation of violence period and 2009 leads to substantial decreases in birth weight (75 grams and a ∼40% increased risk of being <2,500 grams) that are exacerbated for mothers of low socioeconomic status (∼120-125 grams). To put these results in context, the magnitude of the birth weight effect is considerably larger than estimates of the positive impact on birth weight of federal nutrition programs such as the Supplemental Nutrition Program for Women, Infants, and Children (WIC) and the Food Stamp Program (FSP) in the United States. Furthermore, amongst lower socioeconomic status families, the adverse effect of exposure in early gestation to the heightened violence over the last few years in Mexico is equal to the positive impact of the large-scale conditional cash program Oportunidades (PROGRESA) on birth outcomes.

2

On the Long Term Effects of the 1918 U.S. Influenza Pandemic

The 1918 Spanish Influenza pandemic has been widely exploited to provide causal estimates of the longer-term impacts of in utero health insults on physical and economic well-being in adulthood since seminal work by Almond (2006). This body of research indicates there are very large, negative impacts of in utero exposure to the influenza pandemic on health and socioeconomic success in adulthood. The work is widely cited and has been very influential.

Essentially, these studies compare adult outcomes of the 1919 birth cohort, whose mothers had the highest probability of being exposed to influenza during the pregnancy, with comparable children who were not exposed to influenza in utero. Comparisons are drawn between the 1919 birth cohort and those born before and after 1919; a second set of analyses focuses on the 1919 birth cohort and compares those born in areas where maternal mortality rates (MMR) were high with those born in areas where MMR are low. Almond (2006) reports that the exposed cohorts completed significantly less education and earned less as adults than those who were not exposed. The results have been interpreted as powerful evidence that fetal health

has a long-lasting impact, not only on health, but also on economic prosperity in adulthood.

A key assumption underlying this body of research is that the characteristics of the 1919 birth cohort are following the same linear trend as the surrounding birth cohorts. This chapter, using data from the IPUMS samples of the 1920 and 1930 U.S. Censuses, evaluates the validity of this claim. Our results indicate that those who were at highest risk of being exposed in utero were born to families of lower socioeconomic status relative to the cohorts who were not exposed. Specifically, the fathers that produced a child in 1919 were significantly less likely to be WWI veterans, had jobs that produced less income, had lower socioeconomic status (SES), were older, had more total children, and were less likely to be white than fathers of those who were not at high risk of being exposed to influenza in utero.

In an effort to assess the importance of these differences, models of the association between exposure risks and adult outcomes are estimated conditioning on childhood environment. These conditional estimates indicate that the effect of in utero exposure to the pandemic on adult economic prosperity is small in magnitude and not statistically significant. These results suggest that further evidence is required in order to claim that in utero exposure to the influenza pandemic had a persistent impact on a long term outcomes.

2.1 Using the 1918 U.S. Influenza Pandemic to Evaluate the Fetal-Origins Hypothesis

For many decades it has been an accepted fact that what happens during several crucial periods of human development have long lasting effects (Rasmussen, 2001). What has been in dispute over this time, though, is how early these periods begin and how far their impacts span. At the tail end of the 1980's David J. P. Barker introduced what would later be popularly referred to as the fetal-origins hypothesis

(FOH). He suggested that poor health as early as the fetal period had dire consequences for mid to late life chronic diseases (Barker, 1994). Based on sound biological mechanisms and results from animal experiments, this theory has gained a great deal of traction in the medical and social science communities. Moreover, due to the fact that many researchers have linked health with economic outcomes, there is reason to speculate that it may also be the case that in utero health has long term effects on adult SES.

This theory, though, is far more difficult to prove than the original FOH. First of all, a clear biological model and/or an established epidemiological literature does not exist. Secondly, the scope for a behavior response such as directed intervention after birth, while likely ineffective, for example, in the case of arteries that are preprogrammed to harden, seems more promising when considering non-health human capital development. This research question is further complicated by the fact that there are numerous common factors that can jointly and independently determine both in utero health and later life economic well-being (e.g. SES of parents, overall health of parents, quality of caregiving, parents preferences for human capital investment in children). As such, evidence of the link between in utero health and adult economic outcomes, must come from studies that are able to disentangle the intrinsic endogeneity between early-life health and later life SES. By innovatively using the 1918 U.S. influenza pandemic as a natural experiment to assess the long-term effects of in utero health on a large, representative population, Douglas Almond's work became the seminal piece of evidence that the FOH extended beyond long term health into other human capital outcomes.

The justification for using the 1918 U.S. influenza pandemic as a natural experiment revolves around a few keys aspects of its history. The first, and possibly most crucial element is the onset of the disease; the pandemic began unexpectedly in Oc-

tober 1918.[1] This creates the necessary criteria that subjects are unable to change behavior prior to the exposure period in a way that would affect the researcher's sample or group assignment. Further, the disease struck violently, yet quickly, and was almost completely inert by the end of January 1919.[2] In fact, the disease's impact was so condensed that approximately 85% of all the U.S. influenza deaths occurred between October 1918 and January 1918 (Almond, 2006).

The swift onset and departure of the disease also is a useful element as it allows the researcher to assume that there is very little room for meaningful behavior adjustment during the exposure period. Additionally, the pandemic struck an incredibly large portion of the population, 28%, and unlike previous influenza pandemics, this one had particularly high incidence amongst pregnant women and women of childbearing age. This factor allows Almond to treat the the entire 1919 birth cohort as an "intent-to-treat" exposure group (Jordan, 1927, as cited in, Almond, 2006). Moreover, mortality, though severe in terms of typical influenza exposure, was very low, and thus the concern that selective mortality will hinder the accuracy of the estimates is limited.

Finally, the disease is portrayed as having no prejudices. Avoiding the disease was nearly impossible as it was transmitted and obtained through the common air everyone shares. As the old children's rhyme popular at the time explained, "I opened up the window and in-flu-Enza" (Crawford, 2005). Thus, there were extremely variant exposure intensities throughout the country, but most importantly, the heterogeneity in exposure seems to have had no discernible pattern with regard to an area's wealth, climate, or topographical characteristics (Brainerd and Siegler, 2003). In summary,

[1] Most historians now note that the first wave of influenza appeared in March 1918 in an army base in Kansas. This wave though received minimal media coverage at the time and was not reported as influenza until years later, and thus has little potential to impact behavior (Almond, 2006).

[2] There was a final mild flare up of the disease in the spring of 1919, but it was quite benign and went relatively unnoticed and is thus not considered a threat to the validity of the natural experiment (1918.pandemic.gov).

the seemingly ideal methodological construct of the 1918 influenza flu pandemic, created the platform for the most influential analysis to date of the impact of a pregnant mother's health on the later-life outcomes of the child in utero.

Almond (2006) used the 1% sample of the 1960, a combined 3% sample of the 1970, and a 5% sample of the 1980 U.S. Censuses from IPUMS. With this data, he was able to analyze outcomes such as educational attainment, wage and total income, and SES. The primary methodology in this study treats those born in 1919 as the intent-to-treat group and the surrounding birth cohorts, in this case those individuals born between 1912 to 1918 and 1920 to 1922, as the controls. As shown below, his specification measures the effect of being born in 1919, $I_i(YOB = 1919)$, on a later life outcome, y_i, while controlling for the yearly trend, YOB_i, and a quadratic of the yearly trend, YOB_i^2:

$$y_i = \beta_0 + \beta_1 \cdot YOB_i + \beta_2 \cdot YOB_i^2 + \beta_3 \cdot I_i(YOB = 1919) + \epsilon_i \tag{2.1}$$

Table 2.1 presents a replication of Almond's estimates of the coefficient on the 1919 year of birth indicator for regressions run on males in the IPUMS sample of the 1960 U.S. Census. Almost every one of the economic outcomes of interest are statistically significantly adversely affected by being born in 1919. These results are further amplified by the fact that they are based on a group in which only approximately a third of the mothers were infected (Jordan, 1927, cited in, Almond, 2006).

These incredibly stark results have made this work the seminal proof of the connection between maternal health and the long-term future of one's child. In fact, graphs such as Figure 2.1, from Almond's 2006 paper, have become common starting points for policy makers and scientists who would like to stress the importance of fetal programming.

The results from this natural experiment, though, rest on the assumption of

random exposure to the pandemic. Thus, it is critical to investigate the theoretical foundation on which this natural experiment is built, because, while there is no denying the clarity of Figure 2.1, the interpretation of the diagram becomes quite different if exposure status is non-random in a manner correlated with poor later life outcomes.

Figure 2.2, a replication of a similar graph found in Thomas (2010), plots the average socioeconomic status in 1930, as measured by Otis Duncan's socioeconomic index (SEI), of the fathers of people born between 1912 and 1922 by year of birth from the 1930 U.S. Census. This figure, strongly suggests that the 1919 birth cohort, the cohort of interest in Almond's work, had fathers of substantially lower socioeconomic quality. This fact greatly hinders the assumption of randomness necessary for the natural experiment used in Almond 2006. The next section of this chapter will highlight a major event in U.S. history that was taking place during the "exposure" period, describe how the impact of this event may help to clarify the cause of the non-random selection implied by Thomas's figure, and suggest additional characteristics on which the parents of interest may have been selected.

2.2 The Great War and its Implications

The major threat to Almond's natural experiment framework is the fact that overlapping the 1918 U.S. influenza pandemic was an event that significantly impacted fertility during the entire "treatment" period; World War I. Not only is a war of its magnitude always of great demographic significance when evaluating a particular time period, but, in addition, the timing of the United States involvement in WWI is directly correlated with the creation and spread of the 1918 influenza bug.

The United States declared war on Germany in April 1917, was regularly sending troops in the summer of 1918, and had accepted Germany's surrender by November 1918. Thus, during a non-trivial part of the conception period of the exposed cohort

in Almond's study a large and select group of child bearing age men were either stationed in army barracks or overseas and unable to contribute to the production of the 1919 birth cohort. In other words, the 1919 birth cohort is made up of children whose fathers are predominately less likely to have served in WWI. For this selection issue to be a problem, though, it would have to be the case that WWI veterans were, on average and significantly, men of higher parental quality. While in many wars this may be unlikely, there are some legitimate reasons for concern in this case.

First of all, this was the first war in which a U.S. citizen was not allowed to hire a proxy to serve in his place. This ruled out the possibility of the upper class simply buying their way out of service. In fact, due to the draft categories in use in 1917, men with means were more likely to be conscripted. While almost all draft eligible men were put in Class I, one of the main deferments was based on the income dependency of one's family. A man who's family had little financial support apart from himself, such that they would have "insufficient" income if he were drafted, were placed in a lower priority group (Jean Nudd, 2004). Further, as with all drafts, men of particularly low health were either less likely to be drafted or completely removed from the conscription process. These draft classifications suggest a major issue for the assumption of random selection, as the more financially stable and healthy men were more likely to be at war. Thus, it is possible that the 1919 birth cohort is made up of a significantly larger portion of poorer and less healthy families.

Additionally, since the military selection criteria is related to age, men not at war, were likely to be significantly older then the surrounding cohorts. This presents a problem for Almond's strategy as educational cohort trends suggest that younger men were significantly more likely to be literate and educated in this time period. Thus, having an older father meant, on average, having a father with less human capital.

Another avenue through which the war may impact the parental distribution is

through systematic reactions to the experience of living in wartime. Gary Becker has posited a well-known theory of income-driven fertility patterns based on child quantity versus child quality (1960). In essence, he suggests that, like many other durable goods, high-income individuals choose fewer, higher quality children, while low-income individuals choose more, lower quality progeny. This theory offers some intriguing hypotheses when applied to fertility during wartime.

Since, during wartime, families experience more stress, less certainty, and the threat of rationing, parents interested in producing high quality children may wait until the adverse conditions subside. A reasonable hypothesis that follows from this theory is that, during wartime, families with higher income, or at least, families concerned with having higher quality children, may postpone family enlargement until the war is over.

These aspects of life in the U.S. preceding the influenza outbreak suggest that the income, health, and education of the parents of the 1919 birth cohort may have been significantly lower than surrounding birth cohorts and that the exposure cohort families may have had a lower preference for child quality than the comparison cohorts. This type of sorting would present a major problem for identifying the impact of maternal health on the child's later life wealth and education conditions, as numerous studies have connected parental wealth, health, and schooling with these very same outcomes (Hill and Duncan, 1987; Corcoran et al., 1992; Brooks-Gunn and Duncan, 1997; Thomas and Strauss, 1998; Duflo, 2000; Davis-Kean, 2005). In summary, the non-random selection of the draft and the hypothesized non-arbitrary family planning of those experiencing a war, create legitimate concerns over the assumption of random experimental assignment.

While Figure 2.2 implies that the concerns presented previously are real, the goal of the next section of this chapter is to rigorously compare the family characteristics of those born in 1919 with the surrounding birth cohorts. Namely, this study will test

the hypotheses that assert that the parents of children born in 1919 were significantly worse in the areas of income and socioeconomic status, that they were older, and that they desired a larger quantity of, rather than higher quality, children, than the parents of children from surrounding cohorts.[3] The next section will present two approaches to analyzing the validity of these suppositions.

2.3 Methodology

To examine the hypotheses presented in the previous section, it was imperative to find data that contained the parental characteristics of the early 1900's birth cohorts. As Almond, this research takes advantage of the comprehensive and demographically rich U.S. Census data. The IPUMS 1% sample of the 1930 U.S. Census data is particularly useful as it contains information on the parents of U.S. born children over the entire time period of Almond's 2006 analysis, 1912 - 1922. Although the range of parental characteristics is not exhaustive in relation to this study's hypotheses, the 1930 U.S. Census contains ample demographic statistics to provide informative analysis.[4]

One area in which the 1930 census is particularly thorough is in information about the economic status of the parents. The data includes both the father's Duncan's SEI score and the father's occupational income score.[5] Furthermore, family size can be used to address the quantity versus quality hypothesis. In this case, the number of the father's children in the household will be used as a signal of a family's preference.

[3] Unfortunately, this study is unable to directly test the hypothesis that the 1919 birth cohort had significantly less healthy parents as no variable that measured or could proxy for parental health existed in the data. Further, there is no measure of a parent's completed education, thus the Duncan's socioeconomic index, which contains an element of education in its calculation can be scene as the closest proxy.

[4] All data is as of March 31, 1930.

[5] Otis Duncan's SEI is a measure of occupational status based upon the income level and educational attainment associated with each occupation in 1950. Occupational income score assigns each occupation a value representing the median total income (in hundreds of 1950 dollars) of all persons with that particular occupation in 1950.

Another nice element of the 1930 U.S. Census data is that it can be used to directly test the inference that children born in 1919 were less likely to be the child of a WWI veteran. Finally, the age of the father at the time of the child's birth will be used to test if the 1919 birth cohort had significantly older parents than those in surrounding cohorts.

One complication to this study was that the 1930 U.S. Census was collected on April 1, 1930 and age information was obtained as of March 31, 1930. As such, this study is limited to placing people into birth cohort bins between April 1st and March 31st rather than January 1st and December 31st. This hinders the analysis, in that, the birth cohort of interest, 1919, loses an important quarter of exposure, those conceived in the 2nd quarter of 1918, and replaces them with an unexposed group, those conceived in the 2nd quarter of 1919. Following the intuition proposed in the previous section, this would cause the results to be a lower bound, but this issue cannot be tested or solved directly. When discussing the results from the 1930 U.S. Census data, reference to any birth year indicates that the person was born between April 1st of that year and March 31st of the subsequent year.

Additionally, there are two main areas of sampling concern with respect to using the 1930 U.S. Census data for this study. First, we posit that fathers of the 1919 birth cohort were less likely to be in WWI. To be included in the regressions related to a father's characteristics, one's father must be alive in 1930. If it were the case that smarter and more economically viable soldiers were less likely to be killed at war, then the sample of pre-war birth cohort fathers may be biased because the weakest fathers are missing. If this issue is a valid concern, it should be the case that the children born before the war are significantly more likely to be missing data on their fathers. There is no evidence in the data to support this claim.

A second area of concern is that the 1930 U.S. Census does not contain data for one's parents if the person was living independently from their parents. This

is particularly problematic if those children that move out and live by themselves earlier are the children from lower quality households. To determine the severity of this problem this study examined if early birth cohorts, the older children in 1930, had significantly less parental information. In the end, only the earliest birth cohort in the trend, 1912, exhibited this problem. Estimates using a smaller birth cohort group (1913-1922) are qualitatively and quantitatively equivalent to those found in the main analysis.

As this study purposefully follows Almond's own model, the 1919 birth cohort will be isolated to test if it is significantly different than the surrounding cohorts, 1912 to 1922, while controlling for the time trend.[6] The only difference in the two models is that where his outcomes, y_i, were individual i's outcomes in later years, the dependent variables in these specifications are the individual's parent's characteristics in 1930:

$$y_i = \beta_0 + \beta_1 \cdot YOB_i + \beta_2 \cdot YOB_i^2 + \beta_3 \cdot I_i(YOB = 1919) + \epsilon_i \tag{2.2}$$

A second approach to testing the hypotheses of the previous section is to turn to the 1920 U.S. Census. Using the 1920 U.S. Census data provides some straightforward gains. First and most importantly, the 1920 census was taken on January 1st, 1920, thus age perfectly predicts the respondent's year of birth and each birth cohort can be accurately identified. Additionally, due to the fact that the cohorts of interest are 10 years younger in 1920, there is no concern that lower quality older children will have moved out, and as such, left the sample.[7] Along with these beneficial elements of the 1920 census data, though, are some obvious shortcomings.

The major problem with using data obtained on January 1st, 1920 is that the

[6] The actual period used in the analysis was April 1st, 1911 to March 31st, 1923, in order to capture all the respondents born between 1912 and 1922.

[7] As before, the father's data is not missing significantly more for the pre-war cohorts.

comparison group loses almost the entire post pandemic cohort.[8] Although all indications from the 1930 U.S. Census analysis suggest that this is not the case, losing the post pandemic cohort leaves the significant differences found in the 1919 birth group open to the interpretation that they are simply the result of the start of a new trend.[9]

The primary specification will be the same as equation (2.2) except the indicator for being born in 1919 will actually refer to being born between January 1st 1919 and December 31st 1919 and the trend will be from 1912 to 1919.

2.4 Results

Table 2.2 presents the estimates of β_3 from analysis of the IPUMS 1% samples of the 1920 and 1930 U.S. Censuses. Starting with the 1920 U.S. Census results, we see that for both the occupational income score as well as the Duncan SEI outcome, fathers of children born in 1919 are doing significantly worse in 1920, after controlling for the time trend, than the fathers of the previous cohorts. Further, we find that the 1919 birth cohort is a member of significantly larger families, suggesting that Becker's theory of quality versus quantity may be biasing Almond's findings. Additionally, analysis of another marker of parental composition, age of the father at birth, suggests that the fathers of the 1919 birth cohort were significantly older at the time of the child's birth.

Aside from the negative distributional change this suggests with respect to the education of the parents of the 1919 birth cohort, having older parent's may also effect a child's long-term socioeconomic outcomes in an additional way. Having older parents translates into needing to provide care at a younger age. Caregiving which is associated with significantly higher levels of stress (Deimling and Bass 1986;

[8] Only the 4th quarter 1919 birth cohort can be considered relatively unexposed.

[9] In the 1920 U.S. Census they do not ask about military status, so this outcome is not analyzed.

Noelker and Townsend 1987; Stoller and Pugliesi 1989) also may stunt educational and income trajectories, as the time, effort, and money spent on caring for the aging parent can limit the child's ability to take advantage of all opportunities and fully realize their potential.

Finally, the 1920 U.S. Census results reveal that a child born in the U.S. in 1919 was significantly less likely to be Caucasian. This composition change is a clear signal of being born into a less ideal environment as, during this time period, being white provided not just circumstantially better educated and more economically viable parents but, due to rampant racism, also better long term opportunities for one's own achievement.

When evaluating the results for the 1930 U.S. Census we find qualitatively similar results. While the magnitudes of the estimates are smaller due to the loss in precision of the 1919 birth cohort indicator, the direction of the coefficients are always in the hypothesized direction. Finally, as expected, the 1919 birth cohort is significantly less likely to be the child of a World War I veteran. As mentioned, the draft classifications would suggest that children of non-WWI veterans are more likely to be born into financially unstable households. To more firmly establish this claim, we have examined the correlation between being a WWI veteran father and other demographic characteristics while controlling for the father's age, father's age squared, and state of birth fixed effects. For each variable, being a WWI veteran was significantly positively related to having more desirable traits.

Taken as a whole, analysis of the 1920 and 1930 U.S. Censuses indicate that the parents of the 1919 birth cohort were not randomly assigned. Further, the attributes on which they were selected into the "treatment" group are all negatively related to the child's future educational and economic outcomes.

2.5 Re-Evaluation of the Impact of In Utero Exposure to the 1918 Influenza Pandemic on Adult Economic Outcomes

The previous section makes the case that the parents of the 1919 birth cohort were significantly different than the parents of surrounding cohorts in attributes that hinder the identification strategy used in Almond (2006). The next appropriate step to take, after identifying this bias, is to estimate to what extent controlling for parental characteristics reduces the magnitude and significance of Almond's findings. Unfortunately, testing this directly is not possible as the data sources used in Almond (2006) do not contain information on parental or family background characteristics. With this first-best option unavailable we proceed by taking two alternative approaches to estimating the persistent effect of in utero exposure to the 1918 U.S. influenza pandemic when controlling for selection into the 1919 birth cohort.

The first strategy we employed was to replicate Douglas Almond's 2006 work, which uses the 1960, 1970, and 1980 IPUMS samples of the U.S. Censuses, and compare his findings to the same models when they additionally include as close a control for parental characteristics as is available in the data. The most useful data to proxy parental characteristics from the U.S. Census is the information contained in the 1920 and 1930 U.S. Censuses. While we can cannot directly connect an individual record in the later censuses with their parents in the 1920 or 1930 U.S. Censuses, we can apply to each individual the calculated average parental/family information of an individual born in their state of birth, in their year of birth, and of their race from the earlier Censuses.[10]

This analysis was conducted by first replicating Almond's 2006 findings. These estimates are shown in the second column of Table 2.3. Then we next compare the magnitude and significance of the point estimates on the 1919 birth cohort dummy

[10] Race categories were limited to white or non-white.

variable to estimates from the same model that additionally includes state of birth-year of birth-race level parental characteristics from the 1930 U.S. Census. These results are shown in the third column of Table 2.3.[11]

The inclusion of proxies for parental characteristics has a substantial impact on the estimates and implications of Almond's original analysis. Evaluating this exercise one can see that the sign on the coefficients, in all but one case, has reversed. Further, the lone result that has not flipped directions, high school graduation, has been reduced in magnitude by over 75%. Lastly, none of the estimates remain statistically significant at the 5% level.[12] This first approach strongly suggests that accounting for parental characteristics is of first order importance when evaluating the impact of in utero health using the 1918 influenza pandemic as a natural experiment, but as the attributes being used are only proxies, an attempt was made to find data which could both replicate Almond's findings and contained individual level parental characteristics for the cohorts of interest.

The 1973 Occupational Changes in a Generation (OCG) dataset was collected in order to study the importance of a man's background on their adult economic success. As such, it contains information on the subject's employment outcomes as well as on the respondent's family and parental characteristics.[13] The OCG data is made up of a sample of the male 20-65 year old non-institutionalized population in 1973, thus it provides adult outcomes and family characteristics for all of the birth cohorts used in Almond's original study. While the timing of this data does not perfectly match any of the U.S Censuses used in the Almond paper, it is temporally

[11] The 1920 U.S. Census can not be used in this analysis as information is only available for cohorts before 1920.

[12] Conducting the same analysis on the IPUMS samples of the 1970 or 1980 U.S. Censuses provide qualitatively similar results; magnitudes of point estimates are reduced by at least 91% and all statistical significance is lost.

[13] The OCG includes a respondent's parents' education and family income when the respondent was 16 years old and the respondent's number of siblings.

closest to the 1970 census and so this will be used as the comparison to determine if the OCG data can closely replicate Almond's original findings. In Table 2.4, the 2nd and 4th column contain the coefficients from replication of (2.1) when using the highest grade completed by the respondent as the dependent variable and utilizing the 1970 U.S. Census and the OCG data, respectively.

Comparing the two results, the magnitudes of the coefficients are quite similar, with the Census analysis suggesting being born in 1919 leads to a .18 reduction in completed grade level and the OCG estimate implying that being born in 1919 leads to a .16 decrease in completed grade level. The noticeable difference between the two analyses is that the standard error for the Census result is significantly small than the standard error for the OCG analysis. This difference is expected, though, as the Census sample is more than 45 times larger than the OCG sample. To more properly compare the two estimates, we calculate proxy standard errors for the OCG analysis that represent the standard errors of the analysis if the OCG sample size was scaled up to the size of the IPUMS sample of 1970 U.S. Census. This result is seen in row 2 of Table 2.4. Once the OCG data is of comparable size to the 1970 U.S. Census sample, the standard error and thus significance level of the OCG data analysis mirrors very closely what is found in Almond (2006).

Given the similarity of the results, we move forward by introducing the individual level parental and family characteristics to test the impact of controlling for the underlying non-random selection into the 1919 birth cohort. These results can be found in the 5th column of Table 2.4, and, as we found using the first approach, the magnitude of the impact of being born in 1919 is diminished by close to 100%. Even after scaling the OCG sample size to over 300,000, the result is still not statistically significantly different from 0.

Along with the methodology already mentioned, in which only temporal variation is used to identify the impact of fetal health, Almond (2006) contains an alternative

strategy in which adult outcome differences using both temporal *and* geographic variation in influenza exposure are examined. In this analysis Almond uses maternal mortality rates (MMR) by state and the year prior to birth to proxy for infection intensity.

This methodology though, does not control for the identification biases discussed in this study, as high MMRs in one year are likely to be correlated with poor parental characteristics and a weaker health environment for the next birth cohort. High MMRs, particularly when the rate is trending up, can serve as a signal of poor quality health conditions. Moreover, in states where MMRs where relatively high or steadily increasing in the previous year, the families that still choose to conceive a child are likely to have weaker preferences for health. This implies that the fetal health variation Almond is using in this analysis may be significantly correlated with parental and environmental characteristics and, similar to the primary methodology, a failure to control for these factors may lead to biased results. In an effort to test the impact of controlling for these factors, a strategy similar to the first analysis described in this section was conducted in which state of birth-year of birth-race level proxies are generated from U.S. Census data and added to Almond's estimation.

Table 2.5, column 2 contains the results reported in Almond's original 2006 paper.[14] While attempting to replicate this analysis we found a slight error in the MMRs used in the original analysis. It appears that a transcription error lead to assigning Virginia, one of only 19 states with MMR data, a 1919 maternal mortality rate of 6.3 rather than 8.3. Additionally, MMR information for an effected region, Washington D.C., was available in the historic data, and thus added to the analysis.

[14] Two of the reported point estimates, standard errors, and significance levels do not have statistical coherence. While the standard error for the high school graduation regression was reported to be 7.0, from replication we believe the standard error is actually closer to 3.6, making the reported significance level (1% level) in the 2006 paper accurate. The log of total income regression also has an inconsistency in its reported estimates and significance level. In this case, from replication, it appears that the standard error and point estimates are correct but the significance level should be lowered to the 5% level rather than the 1% level.

Replication of Almond's work with these corrections is found in Table 2.5, column 3.

In order to more precisely estimate the impact of influenza exposure on long term economic outcomes using lagged MMRs, we needed to control for two sets of potentially endogenous factors; regional demographic characteristics correlated with MMR but unrelated to the level of influenza exposure and parental attributes of the children born in 1919. Since the MMR analysis is only conducted over the cohorts born between 1918 and 1920, the state of birth-year prior to birth-race level information from the 1920 U.S. Census can be used to control for the demographic factors correlated with a state's MMR in the year prior to birth. Additionally, as in the first analysis mentioned in this section, state of birth-year of birth-race level proxies for family characteristics from the 1930 U.S. Census are added to control for the selection bias inherent in the 1919 birth cohort.

Results from this analysis can be found in Table 2.5, column 4. As before, controlling for parental and environmental factors significantly reduces the magnitude of the point estimates and removes statistical significance in each regression.

While the analysis described in this section is not able to perfectly correct for the identification issues presented in the previous section, each makes a consistent point; the sample selection issue expressed in this study has a significant attenuating effect on the magnitude and power of results that use the 1918 U.S. influenza pandemic as a natural experiment for in utero health and do not control for parental characteristics. Furthermore, these results suggest that influenza exposure in utero was either non-detrimental to long-term economic success, or that some form of intervention on the part of the caregivers of the exposure cohort was effective in remediating the damage to the mechanisms that drive future SES.

2.6 Conclusion

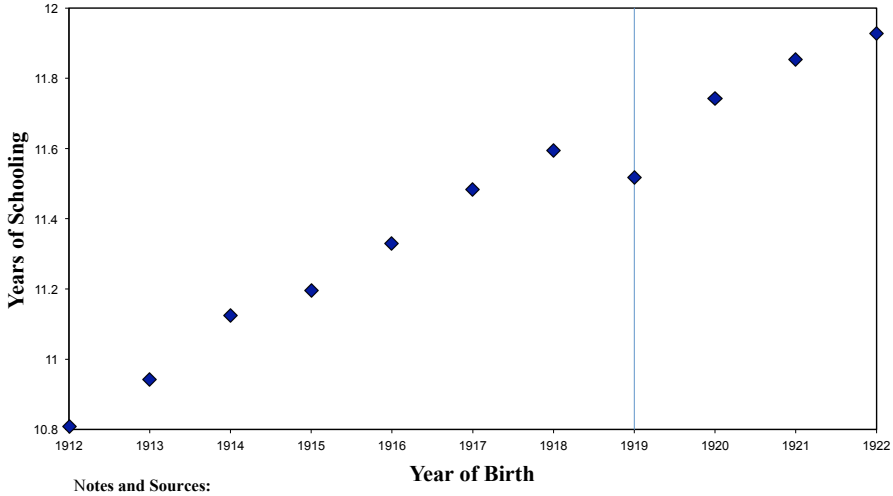
Testing the fetal-origins hypothesis using methods other than a natural experiment is rife with empirical and logistical issues. Controlling for all the typically unobserved parental characteristics correlated with both a parent's health and a child's later life outcomes, as well as, obtaining data which includes the health of pregnant mothers, family characteristics, and follows the child to adulthood is currently not possible for researchers. Given this reality, Douglas Almond's clever use of the 1918 U.S. influenza pandemic and its landmark findings was an incredible breakthrough in the study of fetal health's persistent impact on adult economic outcomes.

This study set out to explore the underlying assumptions necessary to support Almond's influential findings. What we discover is that due to the historical circumstances surrounding the 1918 influenza epidemic, namely WW1's impact on family planning behavior and the systematic selection process of conscription, the 1919 birth cohort was not only exposed to a poorer disease environment in utero, but was also born into families that were significantly less wealthy, larger, and had lower SES.

Most damaging to Almond's inference is that each of these characteristics is a direct or theoretical sign of low quality parentage that can impact a child's later life wealth and educational outcomes. Analysis attempting to replicate Almond's work while adding controls for aspects of the child's family environment consistently return results that suggest that the 1919 birth cohort were not statistically significantly different than surrounding cohorts in their later life education, wages, or SES.

2.7 Tables and Figures

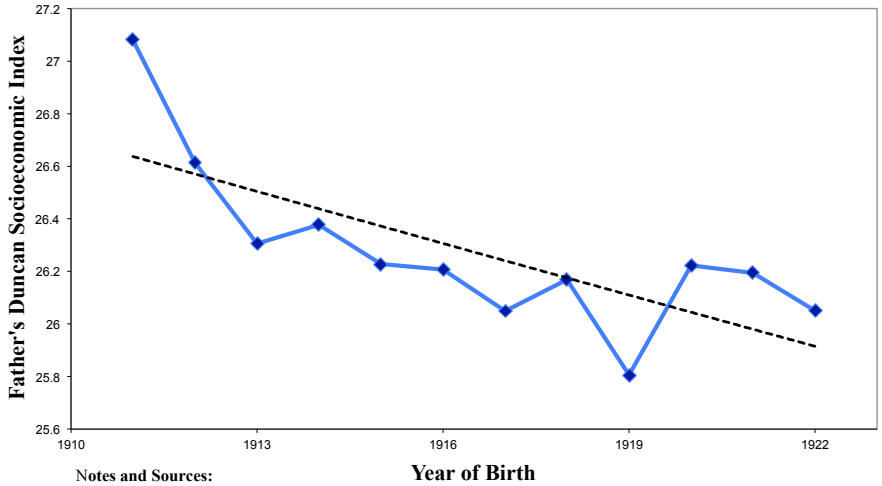
Figure 1



Notes and Sources:
Data obtained from 1 percent sample of 1960 U.S. Census

FIGURE 2.1: Total Years of Schooling by Year of Birth for 1912-1922 Birth Cohorts

Figure 2



Notes and Sources:
Data obtained from 1 percent sample of 1930 U.S. Census
Year of birth covers 4/1/YOB - 3/31/YOB+1

FIGURE 2.2: Father's Socioeconomic Status by Year of Birth for 1911-1922 Birth Cohorts

Table 2.1: Replication of the Almond 2006 Estimates of the Impact of Being Born in 1919 for Men

Long-Term Outcome	Mean	Born in 1919
High School Graduate (%)	47.20%	-2.12% **
		(0.54)
Years of Education (completed)	13.48	-0.15 **
		(0.04)
Total Income ($/month)	5864	-85
		(44)
Wage Income ($/month)	5696	-122 **
		(39)
Poor (% below 1.5 times the poverty level)	27.42%	1.00% *
		(0.49)
Duncan's Socioeconomic Index	35.13	-0.63 *
		(0.26)

Notes:

** indicates statistical significance at the 1% level, * indicates statistical significance at the 5% level.

Regressions use the same models as in Almond (2006) and data from the IPUMS 1% sample of the 1960 U.S. Census.

Robust standard errors are in parenthesis. Regressions based on 114,032 observations.

Table 2.2: Departure of 1919 Birth Cohort Parental Characteristics from Trend[1] for Men

	1920 U.S. Census		1930 U.S. Census
Parental Characteristic	Mean[2]	Born in 1919	Born in 1919[3]
Father's Duncan's Socioeconomic Index	24.39	-1.07 **	-0.23
		(0.36)	(0.22)
Father's Occupation Income Score	22.73	-0.45 *	-0.19
		(0.18)	(0.11)
Number of Father's Children in HH	3.67	0.34 **	0.09 **
		(0.04)	(0.02)
Father's Age at Birth	32.86	0.46 **	0.23 **
		(0.14)	(0.08)
Child is Non-White (%)	11.24%	1.70% **	1.08% **
		(0.53)	(0.33)
Father is a WWI Veteran (%)	6.8%		-1.11% **
			(0.28)

Notes:

** indicates statistical significance at the 1% level, * indicates statistical significance at the 5% level, and robust standard errors are in parenthesis.

Data comes from 1% IPUMS samples of the 1920 and 1930 U.S. Census.

1930 U.S. Census regressions are based on 141,658 observations and 1920 U.S. Census are based on 93,291 observations.

[1]Due to the timing of the 1930 U.S. Census, the trend is from April 1, 1911 to March 31, 1923. For the 1920 analysis, the trend is from January 1, 1912 to December 31, 1919.

[2]Mean for "Father is a WW1 Veteran" comes from 1930 U.S. Census data.

[3]Due to the timing of the 1930 U.S. Census, the 1919 birth cohort consists of people born between April 1, 1919 and March 31, 1920.

Table 2.3: Replication of the Almond 2006 Estimates of the Impact of Being Born in 1919 for Men Compared to Estimates that Control for Parental Characteristics[1]

	Born in 1919	
Long-Term Outcome	Replication	w/ Parent Controls[1]
High School Graduate (%)	-2.1% **	-0.5%
	(0.54)	(0.53)
Years of Education (completed)	-0.15 **	0.01
	(0.04)	(0.04)
Total Income ($/month)	-85	69
	(44)	(43)
Wage Income ($/month)	-122 **	8
	(39)	(38)
Poor (% below 1.5 times the poverty level)	1.00% *	-0.66%
	(0.49)	(0.48)
Duncan's Socioeconomic Index	-0.63 *	0.12
	(0.26)	(0.26)

Notes:

** indicates statistical significance at the 1% level, * indicates statistical significance at the 5% level.

Regressions use the same models as in Almond (2006) and data from the IPUMS 1% sample of the 1960 U.S. Census.

Robust standard errors are in parenthesis. Regressions based on 114,032 observations.

[1]Specification includes birth cohort-state-race level parental characteristics from the IPUMS 1% sample of the 1930 U.S. Census.

Table 2.4: Departure of 1919 Male Birth Cohort From 1912-1922 Trend Using 1973 Occupational Changes in a Generation (OCG) Data

	1970 U.S. Census		1973 Occupational Changes in a Generation		
		Born in 1919		Born in 1919	
Long-Term Outcome	Mean	Baseline	Mean	Baseline	w/ Parent Controls[1]
Years of Education	13.74	-0.18	13.68	-0.16	-0.010
Standard Error Using 1970 Sample Size		(0.02) **		(0.02) **	(0.02)
Standard Error Using OCG Sample Size		-		(0.16)	(0.13)

Notes:

** indicates statistical significance at the 1% level, * indicates statistical significance at the 5% level.

Regressions use the same models as in Almond (2006) and data from an IPUMS combined 3% sample of the 1970 U.S. Census and the 1973 Occupational Changes in a Generation Data. Robust standard errors are in parenthesis.

1970 U.S. Census regressions are based on 308,785 observations and OCG regressions are based on 6,852 observations.

[1]Regressions included individual level parental characteristics.

Table 2.5: The Impact of the Previous Year's Estimated Maternal Infection Rate on Men Born from 1918 to 1920

Long-Term Outcome	Almond (2006)	Corrected[1]	w/Parental Controls[2]
High School Graduate (%)[3]	-10.10% **	-8.64% *	-5.38%
	(7.00)	(4.12)	(4.56)
Years of Education (completed)	-0.756 **	-0.692 *	-0.450
	(0.259)	(0.322)	(0.332)
Log of Total Income[4]	-0.165 **	-0.166	-0.070
	(0.072)	(0.091)	(0.107)
Poor (% below 1.5 times the poverty level)	4.24%	3.17%	-1.27%
	(2.59)	(3.26)	(4.48)
Duncan's Socioeconomic Index	-2.71	-2.39	0.24
	(1.74)	(2.04)	(2.38)
Observations[5]	16,566	16,659	16,659

Notes:

** indicates statistical significance at the 1% level, * indicates statistical significance at the 5% level.

Regressions use the same models as in Almond (2006) and data is from the IPUMS 1% sample of the 1960 U.S. Census.

Standard errors clustered at the state and year of birth level are in parenthesis.

[1]In Almond (2006) there is an error in the Virginia 1919 maternal mortality rate. The error is fixed in this analysis (6.3 changed to 8.3). Additionally, in Almond (2006), District of Colombia births are excluded, but maternal mortality rate information is available for this region.

[2]Specification includes lagged birth cohort-state-race level parental characteristics from the IPUMS 1% sample of the 1920 U.S. Census and contemporaneous birth cohort-state-race level parental characteristics from the IPUMS 1% sample of the 1930 U.S. Census.

[3]While the standard error for the high school graduation regression was reported to be 7.0, from replication we believe the standard error is actually closer to 3.6, making the reported significance level (1% level) in Almond (2006) accurate.

[4]When replicating the total income analysis, it appears that the standard error and point estimates are correct but the significance level should be lowered to the 5% level rather than the 1% level reported in Almond (2006).

[5]This is the total number of observations available, but due to the varying number of missing values for each dependent variable, the total is not the same for each regression.

3

The Intergenerational Impact of Terror: Does the 9/11 Tragedy Reverberate into the Outcomes of the Next Generation?

The September 11, 2001 tragedies in New York City, Arlington, VA and Shanksville, PA extinguished nearly 3,000 lives and shook the United States sense of national security to its core. The unanticipated nature of the attacks along with the devastating imagery of the event produced high levels of psychological distress throughout the nation (Schuster et al., 2001; Knudsen et al., 2005). This wave of stress was persistent, with many experiencing elevated levels for several weeks to months after the attacks, and weighed particularly heavily on women (Silver et al., 2002; Stein et al., 2004). In addition, as suggested in Becker and Rubinstein's theory of responses to terrorism (2011), the fear generated by the event was not limited to those in assaulted areas. In a nationally representative survey Schuster et al. found over 40% of adults reported stress related symptoms after the September 11th attacks. (2001). One particularly troubling aspect of this widespread "terror" shock, is that it may cause the impact of the 9/11 event to spread into the next generation.

Using theoretical models, animal experiments, and small sample human research

the medical literature has biologically mechanized and repeatedly correlated maternal stress with, among other birth outcomes, restricted intrauterine growth and shortened gestational length (de Catanzaro and Macniven, 1992; Wadhwa et al., 1993, 2001, and 2004; Mulder et al., 2002 provides a review). Further, recent and consistent findings have connected birth outcomes to later life human capital accumulation (Behrman and Rosenzweig, 2004; Case et al., 2005; Black et al., 2007). These two lines of research have motivated social scientists to reassess the full negative effect on society of psychologically distressing events such as, discrimination, violence, and natural disasters, by evaluating their impact on the birth outcomes of the exposed pregnant women. This study will add to this emerging literature by using the September 11th, 2001 tragedy as an exogenous stress shock to estimate the response in birth outcomes from the psychological fallout caused by terrorism.

The factors that set this work apart from previous studies of stress and birth outcomes is that it relies on an event with unique attributes that facilitate the precision of the analysis, as well as, utilizes a large and demographically robust dataset. The first element which makes this event particularly suitable to this study is its unanticipated nature. Due to the fact that the stress shock was unexpected, fear of omitted variable bias, a problem faced by many quasi-experimental analyses that struggle to control unobserved factors correlated with maternal stress and maternal qualities that effect birth outcomes, can be greatly reduced. The strategy of minimizing the potential for endogenous non-random maternal characteristic differences in cohorts through the use of an unanticipated event, though, is not a methodology without further complications.

When using an event catastrophic enough to cause significantly elevated stress levels as an experiment, there is significant potential that the tragedy also caused other negative externalities that the exposed will have to endure and may impact the outcome under study. In the case of the September 11, 2001 attacks there are

a number of potential non-stress related shocks which may also effect an in utero child's birth outcomes. While most studies of this event focus on the areas directly effected by the attack New York City (NYC) and the Washington D.C. primary metropolitan statistical area (DC), these are the regions particularly vulnerable to misallocation of stress as the sole contributor to poor birth outcomes. Specifically, it is those cohorts from NYC and DC that, post-attack, are more likely to have mothers that also faced a pollution related adverse health shock and/or a negative resource shock due to loss of economic activity (Bram, Orr and Rapaport, 2002; Landrigan et al., 2004).

Furthermore, when using a tragic event as a natural experiment, it is possible that the assumption of a random treatment group may lose reliability if there is potential for selective migration out of the study area. If a study of the impact of terror on birth outcomes restricts their sample to only those individuals residing in and giving birth in the city that experienced the attack, it must be able to properly control for the group of mothers whose preference for safety and health lead them to move out of the city after the event and thus leave the sample. To date, no large sample study of the September 11th event using NYC and/or DC residents has addressed this problem.

To mitigate concerns over these two sets of identification issues this analysis will exclude cohorts born in NYC and DC. This choice is made because those living in the rest of the country will have had fewer potential negative aftereffects beyond increased maternal stress and, by using the entire country as the sample area, migration concerns are limited.

Finally, an issue that must be carefully considered in all natural experiment studies of an event's impact on in utero health, is that of selective fertility. When using the 9/11 attacks as the event of interest it is fairly straightforward to argue that all cohorts conceived before September 11th, 2001 are randomly assigned to the

treatment or control group, but many studies of this event also use cohorts conceived after September 11th as controls. This is a concerning though, as it is quite plausible that family planning decisions made after the catastrophe could be endogenously related to parental characteristics correlated with birth outcomes. Specifically, this study suggests that cohorts conceived post event have mothers that were significantly more educated and less likely to be African American. This indicates that part of the reference group, in a study that includes post-event cohorts, is non-randomly and positively selected and thus severely hinders the identification strategy. As such, in this study, specific attention is paid to attempting to only analyze cohorts conceived before September 11th 2001.

To this end, using the Vital Statistics Natality Birth Data, which includes all U.S. live births that received a birth certificate, enables analysis to be restricted to births of residents outside NYC and DC and conceived before September 11, 2001, while maintaining very large sample sizes. In addition to the robust sample size, the Vital Statistics data provides this analysis with information imperative to the study such as: demographic characteristics of the mother, birth timing down to the month, and several birth outcomes.

Using this natural experiment framework and detailed data, results indicate that infants in utero during the 9/11 attacks are significantly smaller (5-15 grams smaller and .3% to .4% more likely to be born weighing less than 2,500 grams out of a population with a mean of 7% low birth weight births) and more likely to be born preterm (1% to .4% more likely to be <37 gestational weeks in a population with a mean preterm birth rate of 11%). Further, intrauterine growth is found to be most sensitive to stress exposure in the first trimester and gestational age is most reactive to exposure in mid pregnancy. These findings are consistent with the current medical literature in that they suggest maternal anxiety has statistically significant negative impacts on birth outcomes.

3.1 Literature Review

3.1.1 Stress and Birth Outcomes: Biological Mechanisms

While the physiological level of response each individual has to a stressful event varies, there are certain biological feedbacks which all humans use to regulate psychological distress. In particular, the body unleashes cortisol, norepinephrine, and epinephrine in elevated levels in reaction to acute stress as well as "worry, anxiety, and cognitive preparation for a threat" (McEwen, 1998). These chemicals than stimulate the supply of corticotropin-releasing hormone (CRH). Linking maternal stress to birth outcomes, various studies have indicated that the level of CRH is strongly related to intrauterine growth and parturition timing (Wadhwa et al., 1993, 2004; Mancuso et al., 2004 and others). Additionally, Mulder et al. suggest that arousal of the sympathetic nervous system, a symptom of increased stress, can cause restricted blood flow to the fetus and result in decreased intrauterine growth (2002). Some research has also indicated that the timing of the stress exposure has first order implications on the magnitude of the negative effect.

Multiple medical studies have shown that the release of the hormones associated with a reaction to stress is attenuated during pregnancy and this chemical insulation increases throughout pregnancy (Schulte et al., 1990; de Weerth and Buitelaar, 2005). While this suggests that the adverse effects of maternal psychological distress on birth outcomes should be most prevalent in early gestation, not all studies have supported this claim, and some have even come to the opposite conclusion (Hedegaard et al., 1993; Schneider et al., 1999). As it stands, the medical literature advocates that the timing of in utero stress exposure is important to the biological path of birth outcome damage, but the specific pattern is still without strong empirical support.

3.1.2 Birth Outcomes' Impact on Later Life Health and Human Capital

While generating a clear causal link has been difficult, a growing literature has been building a consensus that health as early as birth can have significant consequences for later life economic, educational, and health outcomes (Strauss and Thomas, 2007, provide an overview of the current literature). Moreover, a set of studies has linked a specific birth outcome, birthweight, to longrun health and human capital accumulation. Of these studies, the work utilizing birthweight differences in twins to control for unobserved parental heterogeneity has generated the most robust findings.

Behrman and Rosenzweig (BR) used data from the Minnesota Twin Registry to conduct an analysis that examined the impact of birthweight differences between monozygotic female twins on their later life health and human capital attainment (2004). They find that more birthweight portends increased height and educational progress. Furthermore, for those at the bottom of the distribution, birthweight differences between twins was predictive of economic wellbeing.

In a more recent study, Black et al. attempt to improve the BR analysis by using a larger set of twins, including both males and females in the analysis, and relying on administrative birth outcome information (2007). With this improved data, Black et al. found results consistent with BR. They report that birthweight has a significant impact on long-term height, IQ, earnings, and education outcomes. While these twin studies are unable to control for parental behavioral changes over time, within families, related to birthweight (e.g. compensating low birthweight with extra parental inputs or investing more heavily in the larger twin) the results are highly suggestive of an important link between birth outcomes and later life wellbeing.

3.1.3 Stress and Birth Outcomes: Prior Evidence

Interest in evaluating the impact of maternal stress on birth outcomes is not a new research area. Over the last few decades there have been many non-experimental studies striving to identify the connection (Newton and Hunt, 1984; Hedegaard et al., 1996; Dole et al., 2003, among others). As with many research areas though, the specter of uncontrolled factors correlated with both the explanatory variables and the outcome of interest have hindered these estimates' validity. Specific to this field, most non-experimental methods are unable to control for all the maternal attributes thought to be correlated both with the maternal stress measurement and adverse birth outcomes (e.g. genetics, health, risk, time discounting preferences, and variance and level of own stress assessment). In an effort to clean analysis of these concerns, some studies have turned to the methodological framework of the natural experiment.

One type of stress inducing event that has been used in several of these works is an earthquake. Glynn et al. used the 1994 Northridge, California earthquake as its stress shock (2001). This work suggested that individuals in utero during mother's exposure to the earthquake early in gestation had lower gestational ages. While this work was innovative in its approach, it suffers from very small sample size (40 women), no control of seasonality or preexisting trends between exposure and non-exposure mothers, and a lack of control for factors other than stress contributing to birth outcome differences (other health, income, or environment shocks associated with the earthquake). A more robust extension of this methodological concept is Torche's recent analysis using the 2005 Tarapaca earthquake in Chile (2011).

The 2005 Tarapaca earthquake provided Torche with a very unique event to study the psychological effects of a natural disaster. Two helpful features of this event in terms of this analysis are Chile's strict building codes and that the earthquake's

epicenter was located in a low density areas. These factors provide some evidence that negative health externalities beyond stress were limited. Further, by using the robust data of Chilean birth certificates the author is afforded a demographically rich and large data source. The results from this study support those found in Glynn et al., in that they suggest that acute maternal stress, specifically early in pregnancy, has significant and non-trivial negative consequences for birth outcomes (51 gram reduction in birthweight and 2.6% increase in preterm births). While migration post-earthquake and the inclusion of the after earthquake birth cohort, both of which may be highly selective, can not be completely ruled out as potential confounds, this analysis is a strong piece of evidence linking acute maternal anxiety and birth outcomes. Another important study in this area is Adriana Camacho's work linking an alternative stress event to poor birth outcomes (2008).

By using random landmine explosions in Colombia as exogenous stress shocks, Camacho is able to utilize a novel source of variation in psychological distress to address this research question. Moreover, in addition to using a model which controls for municipality (similar to a U.S. county) level time invariant heterogeneity, she is also able to conduct alternative analysis using mother-fixed effects. Both models offer qualitatively and quantitatively consistent findings; maternal exposure to a landmine explosion in their municipality significantly decreases birthweight by approximately 8 grams.

One drawback to this study is that due to the reliance on quarterly landmine data, proper analysis of the importance of exposure timing is limited. The results suggest that the effect is strongest two quarters before the birth quarter, but without being able to use birth month specifically, this date range falls in between the first and second trimester. As for identification, this paper is very strong and the concerns are confined to possible non-random geographic sorting related to recent landmine explosions and/or selective migration related to landmine and pregnancy timing (the

mother fixed effects model is limited to non-migrant mothers).

In summary, this study along with Torche's findings, make a strong case that acute maternal stress exposure has statistically significant repercussions for birth outcomes, but the pronounced difference in the magnitudes of the effect in the two studies, the lack of temporal precision in the analysis, and the remaining confounding factors leaves room for additional advancements in this field.

3.1.4 September 11th, 2001 and Birth Outcomes: Prior Evidence

In the years following the tragic events of 9/11 many researchers have expressed concern over the possible negative effects the event may have had on in utero children. These studies have focused in three areas; environmental fallout, discrimination, and stress.

Studies have suggested that the destruction of the World Trade Center (WTC) was the most severe environmental catastrophe in the history of NYC (Landrigan, 2001). After the events on September 11th a gigantic plume containing a mixture of numerous hazardous materials hovered and traveled across NYC (Landrigan et al., 2004). Medical research using samples of pregnant women living or working in NYC have found that exposure to pollutants damaged intrauterine growth and triggered an increase in significantly smaller for gestational age children (Landrigan et al., 2004; Perera et al., 2005). These findings indicate that focusing attention on births outside NYC may be a more accurate way to assess avenues in which the attack effected in utero children beyond direct health shocks from pollution. One interesting line of research to that end has looked at how differential treatment and psychological distress of Arab-named women may have lead to poorer birth outcomes.

Diane Lauderdale and El Sayed et al. hypothesized that, post 9/11, Arabic named women would suffer from significant increases in discrimination and that this would negatively effect their birth outcomes (2006 and 2008). While these studies had

very similar data resources and methodologies, the results were quite different. In California, Lauderdale found that children born to Arabic-named women pregnant during 9/11 had a significantly higher likelihood of being low birth weight (LBW, <2,500 grams) and preterm (PTB, <37 weeks of gestation) than comparison children from the previous year and that this did not hold for any other ethnicities. On the other hand, El Sayed et al. found in Michigan that women with Arab American ethnicity who were pregnant during 9/11 were *less* likely to give birth to a LBW or PTB child.

It is difficult to reconcile these conflicting findings other than to speculate that each state had varying levels of discrimination, as well as, different magnitudes and/or selectivity of in/out-migration (not captured by either analysis). Furthermore, while these studies ask a very intriguing question, they are not able to nail down the mechanism through which discrimination would be effecting birth outcomes. While increased stress is one channel, another major pathway could be financial.

For instance, Kaushal et al. found that wages for Arab-Americans declined after the September 11th attacks (2007). Further, family incomes could be negatively impacted through changed preferences for transactions with Arab-American businesses. Thus, while they represent an innovative approach, the discrimination studies have not formed a consensus and are not aimed at identifying the effects of psychological distress specifically.

A host of studies in the medical literature have attempted to make a more clear statement about the effect of September 11th induced maternal stress on birth outcomes. Several studies used small selected samples of New Yorkers who lived close to the WTC (Berkowitz et al., 2003; Lederman et al., 2004). While these results supported a connection between maternal stress and poor birth outcomes, their geographic proximity to the attack confounds the identification strategy with previously mentioned pollution effects. Since these earlier works, there have been a few addi-

tional papers which have attempted to clean some of these concerns through the use of much larger samples which excluded some or all of the environmentally effected areas or allow them to conduct sensitivity tests of this issue.

Melissa Eccleston, whose paper was written concurrent to this study, explores the impact of the September 11 attacks using birth certificate data. She focuses most of her analysis on New York City residents born between 1995 and 2004. She finds that cohorts in their first or second trimester of gestation weighed significantly less and were born significantly earlier than controls. In order to address the issue of the confounding environmental pollution, she also runs regressions separating out the "less" effected boroughs (Staten Island, Queens, and the Bronx), finding that while the magnitudes are reduced (between 2 and 24%) maternal stress continues to display a significant effect on birth outcomes.

This study though, and any other focusing on residents of the attacked areas, are not without important limitations. By using residents from any part of NYC, the analysis faces the prospect of the exposed cohorts experiencing not just aggravated maternal stress but also a negative resource shock. Multiple studies have shown that NYC employees lost a significant number of labor hours and wages over the next few months following the attacks (Bram, Orr and Rapaport, 2002; Dolfman and Wasser, 2004). Intuitively, loss in income for expecting families can lead to reduced health inputs, causing poorer birth outcomes and thus creating an overstatement of the effect of maternal stress.[1] Moreover, in addition to the income shock faced

[1] Eccleston attempts to address this issue by looking at birth outcomes for the cohort born between August and December 2002. By analyzing this group, which was conceived at least 6 weeks after the event, she suggests that she can assess the effect of the economic downturn on birth outcomes independent of maternal stress. Eccleston finds that this cohort does not have significantly worse birth outcomes and concludes that the economic downturn could not be driving her results. This reasoning though, does not account for the fact that family planning after a major terrorist event in one's city and while facing an economic downturn will be highly selective. Analysis of maternal characteristics of post-event conceiving families in NYC indicate that they were significantly less likely to be African American and more likely to complete additional years of education. Given the endogenous and seemingly positive selection in the post-event conception group, Eccleston's

by the NYC "treatment" group, this cohort may also be contaminated by selective migration.

Following a major health threatening event there may be migration out of the effected area by pregnant women trying to insulate themselves from further stressors or other health insults. Additionally, after an attack on a major city, there may be a reaction by financially able individuals to move out of metropolitan areas as they now seem more dangerous. In fact, in Eccleston's study, she presents evidence that mothers of the exposure cohort in NYC are significantly less likely to be white. Additionally, Eccleston points out that migration statistics based on NYC and NY state income tax filings indicate that from 2001 to 2002 NYC experienced more, and higher income, emigration than the rest of NY state. Taken together, these findings strongly suggest that there are likely to be additional characteristics, unobserved in the birth certificate data, which are also significantly correlated with being a NYC treatment group mother and negatively correlated with birth outcomes.

A final concern, relevant for the Eccleston study, is that including cohorts conceived after the September 11th attack can lead to misidentification. As mentioned in footnote 1, post-event cohorts from attacked cities tend to be from families with positively selected characteristics, thus using them as controls biases the results toward making the treatment cohort look like the event had a larger negative effect on birth outcomes than it truly did. As such, while Eccleston is more rigorous than any previous work using NYC residents, it still struggles to generate clean estimates of the effect of maternal stress because NYC residents were both exposed to several negative birth outcome factors and reacted in systematic ways to the event.

The work most in line with the approach found in this chapter was conducted

robustness check no longer provides any alleviation of the concern over bias caused by the resource shock that was concurrent to the maternal stress shock, as the negative effect of the earning loss will be counterbalanced by the positive sample selection. Analysis of the maternal characteristics of NYC post-event conceiving families is conducted using the same method as in Section 3.4, equation (3.3) and can be found in Table 3.18.

by Eskenazi et al. They used birth certificate data for upstate NY residents in the 40 weeks after the event and compared them to those born during the same period in the preceding two years to shield its analysis from some of the concerns raised previously. The results from this analysis indicated that very low birthweight births (VLBW, <1,500 grams) increased in upstate NY around the New Year (2nd trimester exposure) and 8 months after 9/11 (1st trimester exposure), but moderately low birthweight births (1500-<1999 grams) *decreased* for those born in early December. Results for PTB were also mixed as the authors found that late December births were more likely to be moderate PTB (32-<37 weeks), while those exposed late in pregnancy living in upstate NY were significantly *less* likely to have a moderate PTB.

One issue still faced by this study, due to its focus on upstate NY residents, is the contamination of the "treatment" group by composition change brought on by endogenously selected NYC residents moving out of the city following September 11th. Furthermore, upstate NY residents include many daily commuters into NYC, creating the potential for pollution exposure and experience of the economic fallout in NYC to be impacting the sample.

To avoid the difficulty of identifying maternal stress's relation to birth outcomes using residents from cities that were attacked, a few studies have looked elsewhere for confirmation of the link. Smits et al. looked at over 3,000 Dutch infants in utero during and one year after September 11th, 2001 and found that those exposed while in there 2nd and 3rd trimester had significantly smaller birthweight (2006). Further, a study by Endara et al. using a large dataset of infants born to active-duty military families found *no* effect from being in utero during the attacks (2009). Both of these studies though, rely on the use of the post 9/11 conception cohort as the control group and thus lose part of their identification accuracy as fertility rates and parental characteristics have been found to change after catastrophic events (Evans et al., 2010). Further, Rich-Edwards et al. using 1,184 Boston area women estimated

that those pregnant during 9/11 were *less* likely to have a PTB, but a failure to control for time trends may be driving this counter-intuitive result (2005).

Building off of the lessons of the current literature this study hopes to avoid the various challenges of analyzing this subject in order to bring clarity to the question of whether exacerbated maternal mental stress can significantly hinder birth outcomes and thus potentially reverberate into the future of the next generation.

3.2 Data and Methodology

The data used for this study are the 35,809,694 birth certificates for children born between January 1, 1995 and December 31, 2003 collected by the National Center for Health Statistics available in the Vital Statistics Natality Birth Data (VSNB). In addition to providing a large sample, the data contains several birth outcome variables, as well as, demographic and medical data on the mother and the birth.

When determining a birth's exposure to the September 11th attacks, two methods are employed. The first approach estimates conception date as nine months prior to birth date, mirroring what is typically found in the literature when using only birth timing information. In the VSNB birth date data is available down to the month. As such, for births in September of 2001 it can not be determined whether they were exposed or not and thus, as an attempt to err on the side of a non-result, they will be considered part of the control group. Each of the first 8 birth months post-September 2001 are considered exposed and will be analyzed independently to try and pin down how the timing of the stress event impacts birth outcomes. This approach uses all infants delivered before June 1, 2002 in an effort to limit, as much as possible, to children conceived prior to the event.[2]

As is common in the literature, this study will use a linear reduced form model.

[2] As will be seen in Section 3.4, cohorts conceived after the event are from endogenously and possibly positively selected families and thus their inclusion would jeopardize the randomness of the treatment/control designation.

Specifically the model being estimated using this approach is as follows:

$$b_{imjt} = \alpha_0 + Treat_i^{'}\beta + X_{im}^{'}\delta + \gamma_{yrproxy} + \gamma_{month} + \gamma_j + \gamma_{yrproxy,j} + \epsilon_i \qquad (3.1)$$

where b_{imjt} is the birth outcome of interest for individual i, born at date t, to mother m, that resides in state j.

To evaluate the impact of maternal stress on early life health, the birth outcomes tested include overall birthweight, as well as, indicators for LBW and VLBW births. While these outcomes are the standard in the literature, they actually obfuscate the pathway which is driving the poor birth outcome, as birthweight can be caused by both restricted intrauterine growth as well as shortened gestation. In order to more finely focus the analysis on the biological process driving the birth outcome, two additional dependent variables are included.

To strip the birthweight measure of the impact of gestational length, in order to assess anxiety's impact solely on intrauterine growth, a birthweight for gestation age z-score was created and used as an outcome variable.[3] Further, to look at the other part of the birth outcome equation, gestation age, an indicator for PTB was examined. Finally, there is a medical literature that suggests that maternal stress may impact the sex ratio by reducing male births (reviewed in Catalano et al. 2006), as such, an indicator for being a male infant is also evaluated.

In this equation the matrix $Treat_i^{'}$ is 8 indicators of being born in one of the 8 months from October 2001 to May 2002, representing the exposure period. Additionally, the matrix $X_{im}^{'}$ contains controls suggested by the medical literature including mother characteristics (education, race, marital status, age, plurality, and an indicator for diabetes) and birth information (plurality and sex of infant). Due to VSNB's

[3] Birthweight for gestational age z-score is calculated as an infant's birthweight minus the mean birthweight from 1995 to 2000 for that infant's gestational age, all divided by the standard deviation of birthweight from 1995 to 2000 for that infant's gestational age.

large dataset, controlling for many of these variables can be done with great flexibility, rather than linearly or quadratically, which is the general practice in the literature. Thus, indicator variables are used for mother's education (18 levels), mother's age (36 levels including a level for less than 16 years of age and a level for 50 and over), and parity (8 levels including a level for live birth order of 8 and above).

Additionally, since the method of identification is temporal in nature, controlling for time trends non-parametrically is imperative to proper analysis of this event's impact on birth outcomes. This is made a bit more complicated by the fact that the coefficients of interest include month by month indicators for all births in 2002. In order to include time fixed effects without damaging interpretation of the treatment point estimates, the data from 1995 to 2002 was broken up into 6 equal segments of 16 months. Thus, while true birth year fixed effects are not included, these six, 16 month interval fixed effects, $\gamma_{yrproxy}$, will serve as controls for time trends. In addition it is critical in this type of study to control for seasonality in birth outcomes, and thus, month of birth fixed effects, γ_{month} are also included. Further, to account for any unobserved heterogeneity that is time invariant within the mother's residence state, dummies for mother's state of residence are added to the model, γ_j. Finally, to soak up any location specific time trends, fixed effects for the interaction of an observation's 16 month birth interval and mother's state of residence are incorporated into the specification,$\gamma_{yrproxy,j}$.

A second approach used in the analysis will utilize the more informative but less accurate gestational age data. The VSNB contains data on the weeks a child was in gestation. Researchers have argued that gestational age is incomplete and imprecise (Reichman and Hade 2001) and the concerns stem from the fact that the statistic is predominately based on the mother's report, is in a small number of cases adjusted by a clinical estimate, or may be missing all together. In this study gestational age is used with caution and considerable analysis is conducted only using the birth date

information, but given the fact that, to be a first order problem, the bias would have to take a specific pattern related to the timing of September 11th, much of the concern is attenuated.[4]

When using gestational age (in weeks) together with birth month information, a rough approximation for conception week can be estimated. In this study conception week is calculated as the gestational age minus 2 weeks, as conception usually occurs 2 weeks after the last normal menstrual period, divided by 4, subtracted from the birth month, then increased by 12 if the difference is less than 1. Conception year is then either the birth year or the birth year less one if the conception month is larger than the birth month. Since weekly data must be subtracted from monthly data to generate conception week, each conception week covers a range of conception dates. For example, if an infant is born in the first week of a month the conception week generated in the data is correct. If an infant is born in the last week of a month, though, the conception week generated in the data is early by 3 weeks. As such, to make sure to exclude all births conceived after the event, only infants with a calculated conception date of August 14, 2001 or earlier are included.[5]

When using this second approach the model estimated is as follows:

$$b_{imjt} = \alpha_0 + Treat_i^{'}\beta + X_{im}^{'}\delta + \gamma_{yrproxy} + \gamma_{week} + \gamma_j + \gamma_{yrproxy,j} + \epsilon_i \qquad (3.2)$$

where b_{imjt}, $X_{im}^{'}$, and γ_j are the same as in equation (3.1). In (3.2), $Treat_i^{'}$ is a matrix of 8 indicators for each month of conception from January 1, 2001 to August 14, 2001. Further, as in (3.1) a complete set of year fixed effects can not be used,

[4] The most logical way that measurement error of gestational age would be systematically related to September 11th is if pregnant women were less likely to get or delayed prenatal care following the terrorist attack. As seen in analysis in Section 3.4, Table 3.9, this is not the case. This result provides further support that the accuracy of the gestational age data is not directly impacted by the natural experiment event and thus estimates that take advantage of this information will not be biased by its use.

[5] See footnote 2.

so the data is placed into six equal 16 month groups based on conception date, $\gamma_{yrproxy}$. Finally, since the data contains gestational date by week, the seasonality fixed effects, γ_{week}, are indicators for week of conception. The rest of the controls found in equation (3.1) remain the same.

3.3 Results

Each row of Table 3.1 represents a separate regression and provides the estimates of the β coefficients when using the first approach in which only birth month information is utilized and NYC and DC residents are excluded.[6] The results show that the majority of the significant birthweight effects from maternal distress are grouped in the first trimester of exposure, as cohorts born between March and May 2002 are born significantly smaller, even after controlling for their gestation age, and are more likely to be LBW or VLBW. The timing of stress's effect on gestational age, though, does not exhibit a clear relationship as it is cohorts exposed in the 3rd and 6th months of gestation that are significantly more likely to be born as a PTB infant. On the other hand, while intrauterine growth and gestational age appear to be impacted by acute maternal stress insults, the sex ratio seem to be unaffected.

Similarly, Table 3.2 contains the results of analysis using conception date information, which allows the sample to be stripped more thoroughly of post-exposure conceived infants and gives a more precise estimation of gestational timing of the event. As in Table 3.1, Table 3.2 excludes all NYC and DC residents. As expected, these results are larger and stronger in terms of magnitude and significance. The estimates indicate that almost the entire cohort of children in utero during the attacks had significantly reduced birthweight, by as much as 15 grams. Furthermore, as in

[6] In all tables using the individual level data, robust standard errors are reported and results that are significant using the Schwarz criteria are boxed. The Schwarz criteria is a Bayesian approach to hypothesis testing and is included because it provides a stricter interpretation of statistical significance. In particular, it requires the significance level to be inversely related to sample size: critical t is calculated as the square root of the natural log of n (Schwarz, 1978).

the previous tables, this reduction in birthweight exists for those at the bottom end of the distribution as well; the exposure group children were significantly more likely to be a LBW or VLBW infant.

The estimates in Table 3.2, in regards to exposure timing's effect on intrauterine growth, reinforce the findings from the first approach, in that, once gestational age is controlled for using the z-score, it is clear that intrauterine growth is only significantly restricted by stress exposure in early gestation. Furthermore, when using the more informative conception date data, it it apparent that parturition timing is most sensitive to maternal anxiety in the middle of pregnancy as those cohorts were significantly more likely to be born preterm.[7] Interestingly, the findings indicate that a child's risk of being born LBW or VLBW is related much more strongly to maternal stress's impact on gestational age then through intrauterine growth restriction. The sex ratio, as before, appears to be unaffected by acute maternal psychological distress.

3.4 Discussion

3.4.1 Alternative Specifications

In order to assess the sensitivity of the main results from Table 3.2, several alternative specifications were examined [8]. Table 3.3 contains results from running the same regression as in equation (3.2) on a slightly more selected sample. There may be reason to believe that the resource shock faced by NYC residents also extended out into the surrounding counties around NYC, as many of the residents of the NY

[7] One counter-intuitive result is the finding that those exposed in the first month of gestation were *less* likely to be born preterm. Results from Section 3.4 provide some evidence that this finding may be driven by positive behavioral changes or composition changes of the mothers in this cohort. This will be addressed again in Section 3.4.

[8] Similar alternative specifications following equation (3.1) have also been conducted. The results from these regressions mirror those presented in this section, in that they are evidence of the robustness of the estimates in Section 3.3. Tables 3.11-3.14 contain the alternative specification results when using equation (3.1).

metropolitan statistical area work in NYC. Thus, the results from Table 3.3 exclude not only residents of NYC and DC but the NYC primary metropolitan statistical area as well. The findings using this smaller sample are almost indecipherably different than the baseline results, indicating the group of residents outside NYC are not driving the results.

In the next two sensitivity tests many additional variables are added to the original specification. Given the large number of independent variables and massive sample size being used, the computation burden for these alternative specifications can be quite substantial. In an effort to speed analysis for these two tests, the data was transformed from individual level data to combined cell data. Specifically, the data was collapsed such that each cell contains all the individuals from the same county of residence, week of gestation, year of conception, and sex. Each of the variables of interest are calculated as the mean value for each cell group and the regressions are weighted by the number of individuals that make up each cell. Table 3.4 is a replication of Table 3.2 using this new cell data. The coefficient estimates in Table 3.4 are only negligibly different than those found in Table 3.2 and the interpretation of the impact of maternal stress is unchanged. As such, the results from sensitivity tests using the cell level data will not be driven by the change in the form of the database.

While there are studies that have shown there is a loss in job hours and earnings in NYC after 9/11, it is also quite possible that resource shocks from 9/11 may have differentially and significantly impacted areas all over the country. In order to address this concern, 15 variables, calculated from the Bureau of Labor Statistics, Local Area Unemployment Statistics, where added to equation (3.2) that indicate the unemployment level in the child's county of residence during the 15 months following the estimated conception date. These added controls can proxy for possible economic

fluctuations faced by each child's parents during and following the gestation period.[9] Results from this analysis can be found in Table 3.5. While many of the point estimates from this analysis are slightly larger in magnitude than in the baseline, overall, the results are qualitatively equivalent to those found in Table 3.4, suggesting that differential economic fluctuations related to the September 11th tragedy are not driving the results.

Finally, since the computational burden is reduced when using the cell level data, an analysis was able to be conducted where the state-level, γ_j, and state-time, $\gamma_{yrproxy,j}$, fixed effects are replaced with county-level and county-time fixed effects. By using this finer level of geographic information any unobserved heterogeneity at the county or county and year proxy level can be swept out of the coefficient estimates of interest. The results from this specification are displayed in Table 3.6, and, as in the rest of this section, the results are only marginally different from the baseline. In all, the alternative specifications provided additional support for the baseline results in terms of magnitudes and the temporal variation of the effect.

3.4.2 Test of Assumptions and Possible Confounds

By using the framework of a natural experiment, this analysis requires several strong assumptions and important sample selection choices. The first selection decision that was made for this study was the choice to exclude NYC and DC residents from the analysis. As mentioned previously, this decision was made by relying on previous studies that indicate that, along with being exposed to the stress of September 11th, these individuals also have a higher likelihood of having been exposed to a pollution and/or resource shock, either of which would be negatively related to birth outcomes and confound the estimation of the effect of psychological distress (Landrigan et al.,

[9] The economic activity from approximately six months after birth is included in case the parents are able to reasonably predict coming economic hardship/prosperity and made earlier adjustments to their consumption that would effect the in utero child.

2004; Perera et al., 2005; Bram, Orr and Rapaport, 2002).

A second sample selection decision made for this analysis is to exclude individuals conceived after the events of September 11th. This choice was made as there is concern that family planning choices may have been significantly altered in the months following the tragic events of 9/11. To take a closer look at this issue, maternal characteristics of non-NYC and DC infants conceived in the first seven months following the terrorist attack are compared to the composition of maternal attributes in the rest of the sample period. The regression used in this analysis is as follows:

$$b_{imjt} = \alpha_0 + \beta \cdot POST + \gamma_{1994} + \ldots + \gamma_{2000} + \gamma_{week} + \gamma_j + \epsilon_i \tag{3.3}$$

where POST is an indicator for being conceived in the first seven months after September 11, 2001, γ_{1994}, ..., γ_{2000} are 7 indicators for being conceived in the years from 1994 to 2000, and γ_{week} and γ_j are the same as in equation (3.2). For this test, b_{imjt} will be three maternal characteristics: an indicator for whether the mother is African American, an indicator of whether the mother attended any college, and a measure of the number of years of school the mother completed As such, β is the coefficient of interest and will indicate whether the mothers of the post-event conceived children are significantly different from those conceived in the first 8 months of 2001.[10] The results of this analysis, found in Table 3.7, make a strong statement that the post-event conceiving parents are significantly different than the parents from the previous cohort[11]. Specifically, the mothers have a statistically significantly different racial composition (they are less likely to be African American) and are statistically significantly more educated (both in overall years of school as well as the likelihood of having attended college). Given the potential bias that can

[10] The seasonality controls are of utmost importance in this analysis since the children are born over different sets of months.

[11] A similar analysis using only the birth month information is found in Table 3.15. The results are qualitatively similar.

be caused by including infants from mothers that are endogenously and, most likely, positively self-selected, the choice to cut the sample at those conceived before the event will help preserve the randomness needed for identification using a natural experiment.

With these sample selection choices made, the final assumption that needs verification is that the treatment and control groups being used are randomly assigned and compositionally equivalent. To test the validity of this assumption an analysis of the maternal characteristics of the treatment group was conducted. The specification used to examine the composition of the treatment mothers compared to the control mothers is as follows:

$$b_{imjt} = \alpha_0 + Treat_i^{'}\beta + \gamma_{yrproxy} + \gamma_{week} + \gamma_j + \gamma_{yrproxy,j} + \epsilon_i \tag{3.4}$$

where $Treat_i^{'}\beta$, $\gamma_{yrproxy}$, γ_{week}, γ_j, and $\gamma_{yrproxy,j}$ are the same as in equation (3.2), but the dependent variables being tested are the 3 variables of maternal characteristics from the previous analysis.[12] Additionally, there may be concern that due to the stress caused by September 11th a disproportionate and non-random number of fetal deaths or abortions may have occurred, changing the representativeness of the sample. To test this concern, using the cell level data mentioned in Subsection 3.4.1, a similar regression to (3.4) was conducted where the dependent variable was the number of live births in each cell.

As can be seen in Table 3.8 there appears to be no racial composition difference between treatment mothers and control mothers. Additionally, in terms of college attendance and years of education, while there are a few significant differences, the positive direction of the differences make it clear that this change is not driving the results.[13] Furthermore, the analysis suggests that the exposure group was not

[12] A similar analysis using only the birth month information is found in Table 3.16. The results are qualitatively similar.

[13] This composition change may be responsible for the counterintuitive positive impact of stress

different, in terms of size, than previous cohorts.

Finally, in order to attribute the poor birth outcomes found in Section 3.3 to the biological mechanisms connecting stress to retarded intrauterine growth and restricted gestational age, it is important to establish that the events of 9/11 did not change the health behaviors of mothers. If, for example, mothers pregnant during the event, began to take on unhealthy behaviors such as increased tobacco consumption or decreased use of prenatal care, this would necessarily alter the interpretation of the results found in Section 3.3. To conduct this analysis equation (3.2) is calculated with maternal behaviors (maternal weight gain, as well as, indicators for whether prenatal care started late or never was used, smoking during pregnancy, and alcohol use during pregnancy) as the dependent variables.[14] As seen in Table 3.9, there does not seem to be any systematic negative behavioral reaction by mothers to being exposed to the September 11th events.

3.4.3 Heterogenous Effects

The main results suggest that increased anxiety amongst pregnant women leads to statistically significantly poorer birth outcomes for their in utero children, but this finding may be hiding larger impacts for important sub-populations. For example, a recent study by Torche and Kleinhaus has found that, maternal exposure to psychological stress while in utero has a much stronger negative impact on female children than male, particularly early in gestation (2011). To explore this issue, estimates were calculated splitting the sample by gender. Table 3.10 rows 1 and 2 contain the results on the impact of acute maternal psychological stress on birthweight for the male and female population, respectively. These findings do not show any clear pat-

on PTB found for the August 2001 conception cohort.

[14] A similar analysis using only the birth month information is found in Table 3.17. The results are qualitatively similar.

tern of one gender being discernibly more sensitive to insults of maternal anxiety.[15]

One possible concern with the approach taken in this study is that, by excluding individuals living in the attacked cities, the analysis will lose much of its exposure intensity. While qualitative research suggests that increased anxiety from the September 11th attacks was felt in a diagnosable magnitude by a non trivial number of individuals throughout the country, the next few estimates look into a few important sub-groups in an attempt to find an upper bound on the impact of anxiety exposure.

A sub population that may have experience higher levels of psychological distress after the 2001 terrorist attacks, are those living in large cities. Individuals living in large cities other than NYC and DC may have experienced a higher exposure to psychological distress as they may have internalized the fact that the areas they lived in were the most likely targets for future attacks. Thus, in an attempt to examine whether the country-wide sample is obscuring some larger effect of anxiety on birth outcomes, analysis was run on those living in cities, other than NYC and DC, with a population over 1,000,000 residents. While analysis focused solely on residents of big cities loses some of the identification clarity of the baseline analysis, as it faces potential issues of selective migration, it should provide some evidence of whether the country-wide analysis is grossly underestimating the impact of anxiety exposure. The third row of Table 3.10 display the estimates of the impact of the anxiety of September 11th on the birthweight of children in utero during the attacks. While most of the point estimates are larger than in the baseline, the results from this analysis do not suggest that the baseline is abstracting from an urban sub-population experiencing substantially larger effects of maternal anxiety on birth outcomes.

Another set of hypotheses may be that more or less educated individuals may

[15] Furthermore, there is no significant change in the number of male or female live births in the exposure cohorts.

be more/less effected psychologically by the attacks or may have more/less ability to insulate their in utero child from the trauma of the event. As such Table 3.10 rows 4 and 5 provide estimates when limiting the sample to mother's with a high school degree and mother's without a high school degree, respectively. Neither of these two subgroups produce results that are qualitatively different from the baseline and, moreover there is no clear pattern to suggest that children of less educated mothers experience larger effects from exposure.

3.5 Conclusion

Using an unfortunate and unanticipated national tragedy and a robust source of data, this study estimates the impact that elevated maternal stress has on birth outcomes. In order to develop a clean identification strategy, residents of the attacked areas, who were exposed to other important health and resource shocks in addition to psychological distress, were excluded from the sample, and analysis was limited to those that had made their fertility decision before the event. The findings of this study suggest that, as predicted by the medical literature, infants exposed in utero to increased maternal stress were born significantly smaller and earlier than previous cohorts. Further, month by month analysis indicates that the timing of the stress insult does lead to important differences in the health outcome of the child as intrauterine growth is most sensitive to stress shocks in the first trimester, while gestational age is most susceptible in mid pregnancy.

3.6 Tables and Figures

Table 3.1: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Birth							
Birth Outcome	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Birthweight	27,830,257	3,320	-0.05	0.33	-4.19 ***	2.57	-0.37	-7.91 ***	-3.32 **	-8.31 ***
			(1.49)	(1.52)	(1.51)	(1.50)	(1.53)	(1.50)	(1.51)	(1.50)
BW for GA Z-Score	27,549,946	0.00	0.00	0.00	0.00	0.00	0.00	-0.01 ***	-0.01 **	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	27,830,257	7.5%	0.00%	0.00%	0.08%	-0.04%	0.02%	0.21% ***	0.05%	0.19% ***
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
VLBW (<1,500 g)	27,830,257	1.4%	0.01%	0.04%	0.05%	0.05%	0.01%	0.09% ***	0.05%	0.07% **
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Preterm (<37 wks)	27,566,306	11.5%	-0.02%	0.11%	0.38% ***	0.06%	0.04%	0.20% **	0.07%	0.11%
			(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Male	27,861,010	51.2%	-0.02%	-0.04%	-0.25%	0.06%	-0.08%	-0.13%	-0.11%	-0.10%
			(0.13)	(0.14)	(0.14)	(0.13)	(0.14)	(0.13)	(0.14)	(0.13)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

Table 3.2: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight	27,552,002	3,323	-1.48	**-5.60*** **	**-9.70*** **	**-7.66*** **	**-10.07*** **	**-6.61*** **	**-8.40*** **	**-15.29*** **
			(1.21)	**(1.21)**	**(1.21)**	**(1.23)**	**(1.21)**	**(1.19)**	**(1.19)**	**(1.72)**
BW for GA Z-Score	27,552,002	0.00	0.00 **	0.00	0.00	0.00	-0.01 ***	**-0.01*** **	**-0.01*** **	**-0.02*** **
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	**(0.00)**	**(0.00)**	**(0.00)**
LBW (<2,500 g)	27,552,002	7.4%	0.09%	0.13% **	**0.26%*** **	**0.38%*** **	**0.27%*** **	0.16% ***	0.19% ***	0.22% ***
			(0.05)	(0.05)	**(0.05)**	**(0.06)**	**(0.05)**	(0.05)	(0.05)	(0.08)
VLBW (<1,500 g)	27,552,002	1.3%	0.04%	0.00%	0.08% ***	**0.16%*** **	**0.19%*** **	0.09% ***	0.07% ***	-0.02%
			(0.03)	(0.03)	(0.03)	**(0.03)**	**(0.03)**	(0.02)	(0.02)	(0.04)
Preterm (<37 wks)	27,568,056	11.3%	-0.08%	**0.51%*** **	**0.98%*** **	**0.67%*** **	**0.35%*** **	0.03%	0.15% **	**-0.49%*** **
			(0.07)	**(0.07)**	**(0.07)**	**(0.07)**	**(0.07)**	(0.07)	(0.07)	**(0.08)**
Male	27,568,056	51.2%	0.09%	-0.10%	0.16%	0.05%	0.03%	-0.05%	-0.04%	0.08%
			(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.15)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.3: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Natality Data on Births Conceived before August 14, 2001
Excluding Residents of the New York City and Washington D.C. Metropolitan Areas[1]

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight	27,415,768	3,323	-1.49	-5.61 ***	-9.87 ***	-7.64 ***	-10.11 ***	-6.70 ***	-8.51 ***	-15.29 ***
			(1.22)	(1.21)	(1.21)	(1.23)	(1.21)	(1.20)	(1.19)	(1.73)
BW for GA Z-Score	27,415,768	0.00	0.00 **	0.00	0.00	0.00	-0.01 ***	-0.01 ***	-0.01 ***	-0.02 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	27,415,768	7.4%	0.09%	0.13% **	0.26% ***	0.38% ***	0.28% ***	0.16% ***	0.19% ***	0.22% ***
			(0.05)	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.05)	(0.08)
VLBW (<1,500 g)	27,415,768	1.3%	0.04%	0.00%	0.09% ***	0.16% ***	0.19% ***	0.09% ***	0.07% ***	-0.02%
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.04)
Preterm (<37 wks)	27,431,784	11.3%	-0.08%	0.51% ***	0.98% ***	0.66% ***	0.35% ***	0.03%	0.16% **	-0.49% ***
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.08)
Male	27,431,784	51.2%	0.10%	-0.11%	0.15%	0.05%	0.04%	-0.05%	-0.03%	0.07%
			(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.15)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis.

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

** Significant at 5 percent, *** Significant at 1 percent, **BOX** Significant using the Schwarz Criteria

[1]The New York City and Washington D.C. metropolitan areas are defined as the New York, NY Primary Metropolitan Statistical Area and the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA), respectively.

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.4: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight	780,888	3,259	-1.09	-5.24 ***	-9.23 ***	-7.13 ***	-9.41 ***	-6.36 ***	-7.99 ***	-14.73 ***
			(1.39)	(1.38)	(1.43)	(1.44)	(1.43)	(1.42)	(1.36)	(2.06)
BW for GA Z-Score	780,888	-0.04	0.00	0.00	0.00	0.00	-0.01 ***	-0.01 ***	-0.01 ***	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	780,888	9.9%	0.07%	0.11% **	0.25% ***	0.37% ***	0.25% ***	0.15% ***	0.18% ***	0.21% **
			(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.09)
VLBW (<1,500 g)	780,888	2.6%	0.03%	-0.01%	0.08% ***	0.15% ***	0.18% ***	0.09% ***	0.06% **	-0.03%
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.04)
Preterm (<37 wks)	781,490	13.5%	-0.09%	0.50% ***	0.97% ***	0.65% ***	0.33% ***	0.03%	0.15%	-0.49% ***
			(0.08)	(0.08)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)	(0.09)
Male	781,490	50.4%	0.14%	-0.04%	0.19%	0.09%	0.03%	-0.05%	-0.03%	0.13%
			(1.71)	(1.71)	(1.70)	(1.69)	(1.68)	(1.67)	(1.67)	(2.22)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, week of gestation, year of conception, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.5: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]
and Controlling for County Level Economic Conditions

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight	780,888	3,259	-0.18	-5.86 ***	-10.21 ***	-7.69 ***	-10.82 ***	-7.71 ***	-11.21 ***	-17.23 ***
			(1.41)	(1.39)	(1.45)	(1.45)	(1.45)	(1.46)	(1.40)	(2.09)
BW for GA Z-Score	780,888	-0.04	0.00 **	0.00	0.00	0.00	-0.01 ***	-0.01 ***	-0.01 ***	-0.02 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	780,888	9.9%	0.04%	0.12% **	0.26% ***	0.36% ***	0.27% ***	0.18% ***	0.26% ***	0.27% ***
			(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.09)
VLBW (<1,500 g)	780,888	2.6%	0.02%	-0.01%	0.07% **	0.15% ***	0.18% ***	0.09% ***	0.08% ***	-0.02%
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.04)
Preterm (<37 wks)	781,490	13.5%	-0.16%	0.50% ***	0.99% ***	0.61% ***	0.35% ***	0.06%	0.27% ***	-0.42% ***
			(0.08)	(0.08)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)	(0.10)
Male	781,490	50.4%	0.12%	-0.05%	0.17%	0.09%	0.00%	-0.06%	-0.06%	0.11%
			(1.73)	(1.73)	(1.72)	(1.70)	(1.70)	(1.70)	(1.70)	(2.25)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, week of gestation, year of conception, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects. Additionally, these regressions contain controls for the unemployment level in the child's county of residence during the 15 months following the estimated conception month, calculated from the Bureau of Labor Statistics, Local Area Unemployment Statistics.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.6: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]
and Including County Level Fixed Effects

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight	780,888	3,259	-1.18	-5.29 ***	-9.22 ***	-7.17 ***	-9.37 ***	-6.30 ***	-7.84 ***	-14.53 ***
			(1.33)	(1.30)	(1.36)	(1.36)	(1.36)	(1.35)	(1.31)	(1.98)
BW for GA Z-Score	780,888	-0.04	0.00 **	0.00	0.00	0.00	-0.01 ***	-0.01 ***	-0.01 ***	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	780,888	9.9%	0.07%	0.11% **	0.24% ***	0.36% ***	0.24% ***	0.15% ***	0.17% ***	0.20% **
			(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.09)
VLBW (<1,500 g)	780,888	2.6%	0.03%	-0.01%	0.07% ***	0.15% ***	0.18% ***	0.09% ***	0.06% **	-0.03%
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.04)
Preterm (<37 wks)	781,490	13.5%	-0.09%	0.50% ***	0.97% ***	0.65% ***	0.31% ***	0.02%	0.13%	-0.51% ***
			(0.08)	(0.08)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)	(0.10)
Male	781,490	50.4%	0.15%	-0.04%	0.22%	0.09%	0.07%	-0.04%	0.00%	0.18%
			(1.71)	(1.72)	(1.70)	(1.69)	(1.68)	(1.68)	(1.67)	(2.22)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, week of gestation, year of conception, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's county of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's county of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.7: Change in Maternal Characteristics for Infants Conceived After the 9/11 Attack[1]

Maternal Characteristic	Observations	Mean	Post-Event Cohort[1]	
Mother is African American	29,821,033	14.2%	-0.19%	***
			(0.03)	
Mother's Years of Education	29,417,747	12.79	0.019	***
			(0.003)	
Mother, Some College	29,417,747	45.6%	0.37%	***
			(0.05)	

Notes:
Data obtained from NCHS. Excludes NYC and Washington D.C. PMSA residents.
Includes all births from January 1, 1995 to December 31, 2003 conceived before March 14th, 2002.
Robust standard errors are in parenthesis with, **, meaning significant at 5 percent,
***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.
Each regression controls for conception year fixed effects, conception week fixed effects,
and mother's state of residence fixed effects.
[1]Considered conceived after event if conception week is after August 14th, 2001.

Table 3.8: Departure of Maternal Attributes of Children In Utero During the 9/11 Attack
Using Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Conception							
Maternal Characteristic	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Mother is African American	27,568,056	14.2%	0.02%	-0.04%	0.12%	-0.04%	0.04%	-0.16% **	-0.06%	-0.04%
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.10)
Mother's Years of Educatior	27,196,339	12.8	0.01	0.00	0.01	0.01	0.01	0.04 ***	0.03 ***	0.04 ***
			(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Mother, Some College	27,196,339	45.5%	0.04%	0.05%	0.12%	0.02%	0.16%	0.62% ***	0.46% ***	0.61% ***
			(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.11)	(0.15)
Number of Live Births[3]	781,490	35.3	0.21	1.39	1.14	0.08	0.34	1.01	1.16	1.37
			(1.14)	(1.14)	(1.14)	(1.12)	(1.14)	(1.15)	(1.17)	(1.43)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects,
and an interaction of the conception cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

[3]This analysis uses the cell level data described in Section V.1.

Table 3.9: Departure of Maternal Behaviors of Children In Utero During the 9/11 Attack
Using Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Conception							
Maternal Behavior	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Late/No Prenatal Care[3]	27,033,176	3.7%	-0.02%	0.02%	-0.06%	-0.03%	-0.04%	-0.07%	-0.10% **	-0.15% ***
			(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.05)
Maternal Weight Gain	22,013,433	30.8	0.02	0.03	0.06	0.08 **	0.15 ***	0.17 ***	0.10 ***	0.01
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.05)
Smoke While Preg.	22,691,092	13.3%	-0.06%	-0.01%	0.05%	-0.03%	-0.12%	-0.19% ***	-0.16% **	-0.18%
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.10)
Alch. Use While Preg.	23,483,669	1.1%	0.00%	0.02%	0.01%	0.02%	0.03%	-0.02%	0.01%	0.02%
			(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

[3]Late/No prenatal care defined as either starting prenatal care in the 3rd trimester or never receiving prenatal care.

Table 3.10: Departure of Birth Outcomes for Subgroups of Children In Utero During the 9/11 Attack
Using Natality Data on Births Conceived before August 14, 2001
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Conception							
Birth Outcome	Observations	Mean	Jan 2001	Feb 2001	Mar 2001	Apr 2001	May 2001	June 2001	July 2001	Aug 2001[2]
Birthweight (Males)	14,093,082	3,378	-0.63	-4.69 ***	-9.89 ***	-7.94 ***	-10.09 ***	-6.03 ***	-8.69 ***	-12.66 ***
			(1.74)	(1.74)	(1.73)	(1.76)	(1.73)	(1.71)	(1.71)	(2.45)
Birthweight (Females)	13,458,920	3,265	-2.36	-6.50 ***	-9.51 ***	-7.40 ***	-10.02 ***	-7.16 ***	-8.04 ***	-18.09 ***
			(1.68)	(1.68)	(1.68)	(1.71)	(1.68)	(1.66)	(1.65)	(2.42)
Birthweight (Large Cities)	1,747,147	3,269	-2.55	-3.93	-16.51 ***	-9.52	-19.47 ***	-11.05 **	-13.02 ***	-3.98
			(4.98)	(4.94)	(4.97)	(4.99)	(5.00)	(5.00)	(5.00)	(7.11)
Birthweight (HS Grad)	21,597,004	3,346	-2.38	-5.78 ***	-9.60 ***	-7.74 ***	-10.17 ***	-6.78 ***	-7.92 ***	-14.47 ***
			(1.37)	(1.37)	(1.36)	(1.38)	(1.36)	(1.33)	(1.33)	(1.93)
Birthweight (Not HS Grad)	5,954,998	3,240	2.05	-5.24 **	-9.97 ***	-6.96 ***	-9.19 ***	-4.96	-9.22 ***	-17.58 ***
			(2.61)	(2.58)	(2.61)	(2.65)	(2.64)	(2.65)	(2.65)	(3.84)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month conception cohort fixed effects, conception week fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the conception cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Concieved week between August 1, 2001 and August 14, 2001.

Table 3.11: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of the New York City and Washington D.C. Metropolitan Areas[1]

			Month of Birth							
Birth Outcome	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Birthweight	27,693,676	3,320	0.15	0.41	-4.17 ***	2.54	-0.33	-7.85 ***	-3.18 **	-8.22 ***
			(1.50)	(1.52)	(1.51)	(1.51)	(1.53)	(1.50)	(1.52)	(1.50)
BW for GA Z-Score	27,413,705	0.00	0.00	0.00	0.00	0.00	0.00	-0.01 ***	-0.01 **	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	27,693,676	7.5%	-0.01%	-0.01%	0.07%	-0.05%	0.02%	0.20% ***	0.04%	0.18% ***
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
VLBW (<1,500 g)	27,693,676	1.4%	0.01%	0.03%	0.05%	0.05%	0.00%	0.08% ***	0.04%	0.06% **
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Preterm (<37 wks)	27,430,025	11.5%	-0.02%	0.10%	0.38% ***	0.05%	0.04%	0.20% **	0.06%	0.10% ***
			(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
Male	27,724,382	51.2%	0.00%	-0.02%	-0.24%	0.06%	-0.07%	-0.11%	-0.10%	-0.09% ***
			(0.13)	(0.14)	(0.14)	(0.13)	(0.14)	(0.13)	(0.14)	(0.13)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis.

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's state of residence fixed effects.

** Significant at 5 percent, *** Significant at 1 percent, **BOX** Significant using the Schwarz Criteria

[1]The New York City and Washington D.C. metropolitan areas are defined as the New York, NY Primary Metropolitan Statistical Area and the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA), respectively.

Table 3.12: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Birth							
Birth Outcome	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Birthweight	329,011	3,218	-0.03	0.76	-3.72	2.89	0.05	-7.31 ***	-2.68	-7.97 ***
			(2.03)	(1.93)	(1.94)	(1.92)	(1.94)	(1.92)	(1.96)	(1.89)
BW for GA Z-Score	280,177	-0.06	0.00	0.00	0.00	0.01	0.00	-0.01 ***	0.00	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	329,011	11.5%	0.00%	-0.01%	0.06%	-0.05%	0.01%	0.18% ***	0.03%	0.18% **
			(0.08)	(0.07)	(0.07)	(0.07)	(0.08)	(0.07)	(0.07)	(0.07)
VLBW (<1,500 g)	329,011	3.5%	0.01%	0.03%	0.04%	0.05%	0.00%	0.07% **	0.04%	0.06%
			(0.03)	(0.04)	(0.04)	(0.03)	(0.03)	(0.03)	(0.04)	(0.03)
Preterm (<37 wks)	280,509	15.4%	-0.03%	0.09%	0.35% ***	0.05%	0.03%	0.17%	0.05%	0.10%
			(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Male	336,317	50.5%	0.05%	-0.01%	-0.13%	0.03%	-0.04%	-0.17%	-0.10%	-0.12%
			(4.24)	(4.24)	(4.24)	(4.20)	(4.19)	(4.18)	(4.18)	(4.17)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, month of birth, year of birth, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

Table 3.13: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]
and Controlling for County Level Economic Conditions

			Month of Birth							
Birth Outcome	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Birthweight	329,011	3,218	-0.13	0.46	-4.83 **	1.56	-1.85	-9.02 ***	-5.31 ***	-10.36 ***
			(2.06)	(1.95)	(1.97)	(1.94)	(1.97)	(1.95)	(2.00)	(1.91)
BW for GA Z-Score	280,177	-0.06	0.00	0.00	0.00	0.00	-0.01	-0.01 ***	-0.01 ***	-0.02 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	329,011	11.5%	0.01%	-0.02%	0.08%	-0.02%	0.05%	0.21% ***	0.08%	0.23% ***
			(0.08)	(0.08)	(0.08)	(0.07)	(0.08)	(0.07)	(0.07)	(0.07)
VLBW (<1,500 g)	329,011	3.5%	0.01%	0.03%	0.05%	0.06%	0.01%	0.07% **	0.04%	0.07%
			(0.04)	(0.04)	(0.04)	(0.03)	(0.03)	(0.04)	(0.04)	(0.03)
Preterm (<37 wks)	280,509	15.4%	-0.04%	0.06%	0.38% ***	0.06%	0.06%	0.20% **	0.09%	0.14%
			(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
Male	336,317	50.5%	0.01%	-0.05%	-0.15%	-0.02%	-0.05%	-0.25%	-0.15%	-0.19%
			(4.28)	(4.27)	(4.28)	(4.23)	(4.23)	(4.23)	(4.24)	(4.21)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, month of birth, year of birth, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's state of residence fixed effects. Additionally, these regressions contain controls for the unemployment level in the child's county of residence during the 15 months following the estimated conception month, calculated from the Bureau of Labor Statistics, Local Area Unemployment Statistics.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

Table 3.14: Departure of Birth Outcomes for Children In Utero During the 9/11 Attack
Using Collapsed Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]
and Including County Level Fixed Effects

			Month of Birth							
Birth Outcome	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Birthweight	329,011	3,218	-0.27	0.45	-3.79 **	2.47	-0.28	-7.47 ***	-3.01	-8.13 ***
			(1.68)	(1.60)	(1.60)	(1.63)	(1.67)	(1.62)	(1.67)	(1.65)
BW for GA Z-Score	280,177	-0.06	0.00	0.00	0.00	0.01	0.00	-0.01 ***	0.00	-0.01 ***
			(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
LBW (<2,500 g)	329,011	11.5%	0.01%	-0.01%	0.06%	-0.04%	0.02%	0.19% ***	0.04%	0.18% ***
			(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
VLBW (<1,500 g)	329,011	3.5%	0.01%	0.03%	0.04%	0.05%	0.00%	0.07% **	0.04%	0.06%
			(0.03)	(0.04)	(0.04)	(0.03)	(0.03)	(0.03)	(0.04)	(0.03)
Preterm (<37 wks)	280,509	15.4%	-0.01%	0.10%	0.36% ***	0.06%	0.04%	0.17%	0.05%	0.09%
			(0.09)	(0.09)	(0.09)	(0.09)	(0.10)	(0.09)	(0.09)	(0.09)
Male	336,317	50.5%	-0.01%	-0.04%	-0.11%	-0.03%	-0.09%	-0.21%	-0.17%	-0.13%
			(4.25)	(4.25)	(4.25)	(4.21)	(4.20)	(4.19)	(4.19)	(4.18)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates. Data was collapsed such that each cell contains all the individuals from the same county of residence, month of birth, year of birth, and sex. The regressions are weighted by the number of individuals that are contained in each cell.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent and, ***, meaning significant at 1 percent

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's county of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's county of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

Table 3.15: Change in Maternal Characteristics for Infants Conceived After the 9/11 Attack[1] Using Birth Date Information Only

Maternal Characteristic	Observations	Mean	Post-Event Cohort[1]	
Mother is African American	30,137,299	14.2%	-0.18%	***
			(0.03)	
Mother's Years of Education	29,707,280	12.78	0.028	***
			(0.002)	
Mother, Some College	29,707,280	45.5%	0.52%	***
			(0.04)	

Notes:
Data obtained from NCHS. Excludes NYC and Washington D.C. PMSA residents.
Includes all births from January 1, 1995 to December 31, 2002.
Robust standard errors are in parenthesis with, **, meaning significant at 5 percent,
***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.
Each regression controls for birth year fixed effects, birth month fixed effects,
and mother's state of residence fixed effects.
[1]Considered conceived after event if born after May 31, 2002.

Table 3.16: Departure of Maternal Attributes of Children In Utero During the 9/11 Attack
Using Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Birth							
Maternal Characteristic	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Mother is African American	27,861,010	14.2%	-0.19% **	-0.11%	0.02%	-0.28% ***	-0.20% **	-0.21% **	-0.22% **	-0.29% ***
			(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)
Mother's Years of Education	27,461,455	12.8	0.00	0.00	-0.01	0.02 ***	0.02 **	0.02 **	0.03 ***	0.04 ***
			(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Mother, Some College	27,461,455	45.4%	-0.17%	-0.04%	-0.24%	0.21%	0.11%	0.28% **	0.32% **	0.59% ***
			(0.13)	(0.13)	(0.13)	(0.13)	(0.14)	(0.13)	(0.13)	(0.13)
Number of Live Births[2]	291,590	95.5	0.19	0.91	0.87	1.52	2.92	1.81	4.30	5.04
			(8.30)	(8.12)	(8.18)	(8.16)	(7.96)	(8.21)	(8.15)	(8.30)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, and an interaction of the birth cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]This analysis uses the cell level data described in Tables B2-B4.

Table 3.17: Departure of Maternal Behaviors of Children In Utero During the 9/11 Attack
Using Natality Data on Births from January 1, 1995 to May 31, 2002
Excluding Residents of New York City and the Washington D.C. Metropolitan Area[1]

			Month of Birth							
Maternal Behavior	Observations	Mean	Oct 2001	Nov 2001	Dec 2001	Jan 2002	Feb 2002	Mar 2002	Apr 2002	May 2002
Late/No Prenatal Care[2]	27,248,623	3.8%	0.01%	-0.02%	0.03%	-0.14% ***	-0.11% **	-0.03%	-0.06%	-0.14% ***
			(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)	(0.05)
Maternal Weight Gain	22,048,841	30.8	0.007	0.008	0.055	0.117 ***	0.099 **	0.216 ***	0.063	0.000
			(0.040)	(0.041)	(0.041)	(0.040)	(0.041)	(0.040)	(0.040)	(0.040)
Smoke While Preg.	22,754,458	13.3%	0.04%	-0.02%	0.19% **	-0.02%	0.01%	-0.17%	-0.07%	-0.20% **
			(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)
Alch. Use While Preg.	23,547,147	1.2%	-0.01%	0.02%	0.04%	-0.03%	0.01%	0.02%	0.02%	0.00%
			(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)

Notes:

Data obtained from National Center of Health Statistics 1995 to 2002 birth certificates.

Robust standard errors are in parenthesis with, **, meaning significant at 5 percent, ***, meaning significant at 1 percent, and **BOX** meaning significant using the Schwarz Criteria.

Each regression controls for 16 month birth cohort fixed effects, birth month fixed effects, mother's state of residence fixed effects, fixed effects for live birth order (8-levels), fixed effects for mother's age (36-levels), fixed effects for mother's education level (18-levels), parity, mother's diabetes, mother's race (white, black, Hispanic, and other), child's gender, and an interaction of the birth cohort and mother's state of residence fixed effects.

[1]The Washington D.C. metropolitan area is defined as the Washington, DC-MD-VA-WV Primary Metropolitan Statistical Area (PMSA)

[2]Late/No prenatal care defined as either starting prenatal care in the 3rd trimester or never receiving prenatal care.

Table 3.18: Change in Maternal Characteristics for New York City Infants Conceived After the 9/11 Attack[1]

Maternal Characteristic	Observations	Mean	Post-Event Cohort[1]
Mother is African American	963,731	28.0%	-0.55% ***
			(0.24)
Mother's Years of Education	945,583	12.55	0.028 *
			(0.016)
Mother, Some College	945,583	40.3%	0.10%
			(0.27)

Notes:
Data obtained from NCHS. NYC residents only.
Includes all births from January 1, 1995 to December 31, 2003 conceived before March 14th, 2002.
Robust standard errors are in parenthesis with, *, meaning significant at 10 percent,
, meaning significant at 5 percent, and ,*, meaning significant at 1 percent
Each regression controls for conception year fixed effects, conception week fixed effects,
and mother's state of residence fixed effects.
[1]Considered conceived after event if conception week is after August 14th, 2001.

4

The Mexican Drug War and Early-Life Health: The Impact of Violent Crime on Birth Outcomes

Since 2008, rates of crime and violence in Mexico have risen at a dramatic and unprecedented pace. According to official data reported by the National Institute of Statistics and Geography (INEGI), homicide figures in Mexico had been stable and declining from the mid-1990's until 2007, but between 2007 and 2010 the number of reported murders almost tripled (from 8,845 in 2007 to 25,000 in 2010, Figure 4.1 displays yearly homicide totals and Figure 4.2 provides monthly homicide rates from 2000 - 2011). While the specific causes of this horrific change in the criminal environment are still being debated, what is undeniably clear is that Mexico is suffering one of the most sudden and deadly internal conflicts in recent history.

Given the incredible magnitude of this outbreak of violence many scholars have already begun to examine the short and long-term effects this ongoing tragedy will have on the citizens of Mexico (Dell, 2011; Brown and Velasquez, 2013; Robles et al., 2013, Velasquez, 2013, among others). The purpose of the current study is to add to this growing set of research by rigorously investigating the impact of the rise

in violence on a group of individuals that is particularly vulnerable and, in terms of Mexico's future, very important: infants. Specifically, the goal of this research is to estimate the extent to which a pregnant mother's exposure to violence can restrict human capital accumulation at its earliest stage, in-utero, by examining the impact of the escalation of the Mexican drug war on the birth outcomes of its citizens.

This research question is motivated by several potential mechanisms that may connect maternal experience of crime and conflict to the early-life health of the in-utero child. For one, the magnitude and conspicuous nature of the drug war violence in Mexico has made psychological exposure essentially unavoidable in highly affected areas and maternal mental stress has been associated with intrauterine growth and gestational length restrictions of the exposed in-utero child (Beydoun and Saftlas, 2008; Camacho, 2008; Brown, 2013). Additionally, researchers have found that individuals experiencing increasing levels of violence are suffering poorer economic outcomes (Dell, 2011; Robles et al., 2013; Velasquez, 2013). A loss in family resources driven by local conflict has the potential to hamper the development of the fetus through decreased consumption of nutritious foods and vitamins and/or restricted use of prenatal care.

Discovering the validity and magnitude of the relationship between violence from the Mexican drug war and birth outcomes has increased importance as economists continue to identify a strong and persistent association between birth weight and later life outcomes such as IQ, height, educational attainment, and wages (Behrman and Rosenzweig, 2004; Black et al., 2007; Figlio et al., 2013). Additionally, while much of the early work examined this relationship in developed countries, subsequent research has provided evidence of this link in more diverse settings (Bharadwaj et al., 2010 and Torche and Echevarria, 2011 in Chile and Rosenzweig and Zhang, 2012 in China).

Generating a causal link between the fetal health consequences of exposure to lo-

cal violence, though, presents numerous challenges. For example, differential regional conflict levels may be correlated with pre-existing differences or trends in various local factors that are also correlated with the robustness of the population of mothers. Furthermore, behavioral responses, such as migration and family planning, to rising crime may cause a child's exposure level to be correlated with observed and unobserved characteristics of the mother, which in turn are also related to fetal health. This study relies on the timing, level of detail, and persistent tracking efforts of the Mexican Family Life Survey (MxFLS) to address these concerns in a more rigorous way than any previous study of this topic.

The MxFLS is a nationally representative longitudinal study that has been conducted in Mexico since 2002 and spans both the pre-escalation of violence and escalation periods while maintaining very high levels of survey retention. Along with detailed individual- and household-level economic, health, and migration data, the MxFLS also contains a particularly useful feature for this study: reproductive histories. This module provides information on the birth weight, prenatal care usage, pregnancy complications, and location of delivery for all births since the last interview. This rich survey is then paired with the month and municipality-level homicide data collected by INEGI to generate estimates of the impact of increased local violence in key gestational periods on the birth outcomes of exposed children.

Using this data, which allows for comparison of pre-conflict and conflict period cohorts, control of all time-invariant unobserved heterogeneity at the level of the mother through sibling fixed effects, and removal of endogenous migration through an intent-to-treat approach, this study finds children exposed to local violence while in their first trimester of gestation had a sizable and significant decrease in birth weight (75 grams) and a massive increase in the probability of being designated low birth weight (3-5% out of a base of 7-10%).[1] Moreover, children of lower socioeconomic

[1] These magnitudes were calculated based on the average increase in the 3-month homicide rate

status families suffered even larger adverse outcomes, with the magnitude of the first trimester exposure effect on birth weight doubling in size.

To put these results in context, the magnitude of the birth weight effect is considerably larger than estimates of the positive impact on birth weight of federal nutrition programs such as the Supplemental Nutrition Program for Women, Infants, and Children (WIC) and the Food Stamp Program (FSP) in the United States and are about one-third to two-thirds the size of the adverse consequences of maternal smoking.[2] Furthermore, amongst lower socioeconomic status families the adverse effect of exposure in early gestation to the escalated violence in Mexico is equal to the positive impact of the large-scale conditional cash program *Oportunidades (PROGRESA)* on birth outcomes.[3] Overall, the consistency and scale of these findings suggest that the deleterious effect of the Mexican drug war on the population of Mexico may reverberate for an entire generation.

4.1 Motivation

4.1.1 Organized Crime's Leading Role in Violence in Mexico

The sudden change in the magnitude and subtleties of conflict related to the drug trade in Mexico, as well as, the increasing spillover of violence onto civilian non-actors, has put an international spotlight on the Mexican "war on drugs".

This increased interest has led to an in-depth study of and vigorous debate about

between 2009 and the pre-escalation of violence period of 2005-2007 (.25 additional homicides per 10,000 inhabitants).

[2] Hoynes et al. estimate a 2 gram effect of WIC on the average population and a 18-29 gram impact amongst participants (2011). With regard to FSP, Almond et al. suggest the program led to birth weight increases of 2-5 grams in general and 15-40 grams amongst the treated (2011). There have been many types of studies of maternal smoking's effect on birth weight with a consensus forming around a magnitude of about 200-230 grams and 100-130 grams for heavy (11+ cigarettes a day) and light smokers, respectively (Rosenzweig and Shultz, 1983; Sexton and Hebel, 1984; Brooke et al., 1989; Wilcox, 1993; Almond et al., 2005; Ward et al., 2007).

[3] Barber and Gertler estimate a 127 gram increase in the birth weight of children born to mothers participating in *Oportunidades* (2008).

its causes (Guerrero, 2011; Rios and Shirk, 2012; Robles et al., 2013, provide excellent holistic descriptions of the conflict's history and actors). The general consensus though, with varying opinions to the magnitude of each factor, is that the spike in homicides is a byproduct of three interrelated events. One aspect is the increased success of the USA-Colombia fight to reduce the flow of drugs between the two countries, giving Mexican drug cartels extra incentive to control the increasingly profitable drug trade (Castillo et al., 2012).

A second major influence was former president, Felipe Caldern's, strategy of increased federal military opposition to OCGs (Molzahn et al., 2012; Guerrero, 2011). The military approach taken by Caldern was to unilaterally challenge all OCGs, regardless of the size or location of the territory they controlled. As would be expected, this tactic has resulted in increased and geographically dispersed conflict throughout Mexico (Guerrero, 2011).

Lastly, the changes in military policy during the past few years have fostered an unexpected and unintended alteration in the overall picture of crime in Mexico. When the military succeeded in capturing or killing a high-ranking cartel member this would regularly result in intense fighting within the group to fill the power vacuum and eventually the fracturing of the original OCG into several new crime organizations. Guerrero finds that between 2006 and 2010 the number of OCGs grew by a factor of more than 2.5 (2011). The increased number of crime groups operating in a limited space and competing over finite profits has amplified violence between these groups. Moreover, this fighting has changed the conflict environment for non-combatants as the increased use of intimidation and scare tactics through conspicuous violence and criminal activities targeting innocents has not been merely a negative externality of the OCGs actions, but also a targeted agenda goal, as the OCGs seek to reduce the willingness for citizens to mobilize or cooperate with the police or rival cartels.

Another major negative spillover from the war on drugs has been an increase in non-drug related crimes that target non-combatants. As profits from drug running are reduced in size, due to military interference and the need to split the proceeds between more groups, OCGs have increasingly turned to crimes perpetrated on law-abiding citizens such as extortions, kidnappings, and car thefts (Molzahn et al., 2012; Guerrero, 2011).

In summary, the pregnant women in Mexico that form the population of interest for this study faced an environment in the late 2000's that was in stark contrast to the world they had lived in just a few years before. In particular there was a dramatic and larger rise in the potential for physical, mental, and financial harm. This study is interested in analyzing how a rise in this type of potential victimization can hinder the early life health of the next generation.

4.1.2 Conflict and In-Utero Human Capital Development: Pathways

While generating a clear causal link has been difficult, a growing literature has been building a consensus that health as early as birth can have significant consequences for later life economic, educational, and health outcomes (Strauss and Thomas, 2007, as well as, Almond and Currie, 2011 provide overviews of the current literature). Moreover, a set of studies has linked a specific birth outcome, birth weight, to the longrun accumulation of human capital such as height, IQ, earnings, and education (Behrman and Rosenzweig, 2004; Black et al., 2007; Figlio et al., 2013 among others). This link between birth weight and long run economic outcomes suggests that if the Mexican drug war is hindering fetal health, its impact on Mexican society will linger into the next generation. Unfortunately, there are compelling reasons to believe that the violence in Mexico has the potential to harm in-utero health and birth outcomes through several pathways of vulnerability including maternal anxiety, restriction of resources, and reduced access or willingness to utilize prenatal health services.

A growing literature has emerged that rigorously examines the impact of maternal anxiety on the birth outcome of the in-utero child. Using theoretical models, animal experiments, and small sample human studies the medical literature has biologically mechanized and repeatedly correlated maternal stress with, among other birth outcomes, restricted intrauterine growth and shortened gestational length (de Catanzaro and Macniven, 1992; Wadhwa et al., 1993, 2001, and 2004; Mulder et al., 2002 provides a review).

Specifically, one theorized mechanism is that the body produces excess cortisol, norepinephrine, and epinephrine when confronting "worry, anxiety, and cognitive preparation for a threat" (McEwen 1998) and this reaction stimulates the supply of corticotropin-releasing hormone (CRH), which is strongly linked to intrauterine growth and parturition timing (Wadhwa et al., 1993, 2004; Mancuso et al., 2004 and others). An additional channel suggested by Mulder et al. is the stress induced arousal of the sympathetic nervous system, which can cause restricted blood flow to the fetus and result in decreased intrauterine growth (2002). Some research has also indicated that the timing of the stress exposure is paramount in determining its impact on fetal health.

Several studies have posited that as a pregnancy progresses the fetus is less and less at risk to fluctuations in maternal CRH levels because the mother is less reactive and has dampened sensitivity to stressful events (Schulte et al., 1990; de Weerth and Buitelaar, 2005). Furthermore, CHR and cortisol levels naturally increase throughout pregnancy, which may in turn work to insulate the fetus from later term maternal anxiety shocks.

Establishing a causal link with regards to the overall effect of maternal stress as well as the importance of the exposure timing has proven quite challenging. Recently a few studies, relying on natural experiment techniques, have been able to more credibly identify the impact of acute stress from events such as landmine explosions

(Camacho, 2008), terrorist attacks (Brown, 2013), and earthquakes (Torche, 2011). These papers have provided a consistent picture: maternal anxiety exposure in early pregnancy leads to significantly poorer birth outcomes.

This literature as a whole suggests that the anxiety from the fear and victimization that has been all too present in Mexico in the last few years has a direct biological pathway in which it can damage the early life health of the exposed children. Moreover, there are additional mechanisms that are not explicitly related to the impact of increased cortisol on the regulation of the fetus that may link increased fear and anxiety to poor birth outcomes.

Terror and depression induced by exceedingly high levels of realized or potential victimization may cause pregnant mothers to alter their behavior in several harmful ways. The experience of increased stress and loss of control may lead to the escalation of risky behaviors such as smoking and drinking. Additionally, fear may cause women to be less likely to access prenatal health care, which has been associated with the quality of the birth outcome in both developed and developing countries (Rosenzweig and Schultz, 1983, United States; Jewell and Triunfo, 2006, Uruguay; Jewell, 2007, Bolivia, Brazil, Colombia, and Peru; Wehby et al., 2009, Argentina). In addition to mental hardship, these mothers may also be experiencing direct insults to nutrition through a tightened resource constraint.

Recently a few papers have examined the impact of the Mexican outbreak of violence on the income and earnings of the Mexican population (Dell, 2011; Velasquez, 2013; Robles et al., 2013). Each study, using a different identification strategy, has found that the conflict has had a negative impact on the labor market participation and earnings of Mexican workers. If this reduction in financial resources leads to a change in consumption patterns, the health of the in-utero child may suffer. For example, Almond and Mazumder suggest that children exposed during the gestational period to a mother who is restricting their food intake due to observance of Ramadan

are born significantly smaller (2011). Furthermore, if the restricted income potential impacts a family's ability to afford prenatal care, this would further put the fetus's health at risk.

4.1.3 Conflict and Human Capital Development In-Utero: Prior Evidence

What is clear is that there are many reasons to think that violence and conflict may have significant consequences for the birth outcomes of exposed children, yet very few studies have rigorously attempted to identify the magnitude of the effect. While studies of the impact of maternal anxiety have had some success by using plausibly exogenous shocks of psychological stress for identification, these papers do not provide an apt substitute for the study of the impact of crime and victimization on in-utero health as they typically rely on infrequent or short-term events and identify only one of the channels (maternal anxiety) by which conflict may hinder proper fetal development.

Most of the literature that specifically analyzes the impact of conflict on birth outcomes has relied on persistent variation between localities in rates of violence, which may be correlated with other unobserved or omitted factors that differ between the regions and are correlated with early life health. Furthermore, these studies, by using locations with fairly well established and constant levels of violence over time, are subject to conflating systematically taken behavioral responses, such as residential sorting and family planning, with the actual impact of violence on birth outcomes. Recently, one study combined an unexpected outbreak of violence with the identification strength of within family comparisons to produce results, which remove many of the concerns presented in the previous literature.

In 2012, Hani Mansour and Daniel Rees published their work examining conflict and birth weight in Gaza and the West Bank during the al-Aqsa Intifada. The al-Aqsa Intifada started with the contentious visit by Ariel Sharon to the Temple

Mount in September 2000 that sparked 4 months of violence, followed by a reduction in conflict for 8 months, at which point clashes intensified again until the summer of 2002, with some level of residual violence lasting into 2005. Mansour and Rees evaluate the impact of fatalities caused by Israeli troops, at the month and district level, on the birth outcomes of children born between April 2001 and June 2004. They find that an increase in fatalities in the district of birth 9-6 months before birth lead to a statistically significant increase in the probability that the child will be born low birth weight (LBW, <2,500 grams). The strength of their analysis lies in the fact that they can exploit temporal and geographic variation in conflict intensity and, as some mothers had two children between April 2001 and June 2004, they can control for time-invariant characteristics of the mother using sibling fixed effects. This analysis and its identification strategy is a major contribution to the current literature and remains the seminal work in the area of conflict and birth outcomes.

One issue this analysis faces, though, is that all of the data was collected and almost all of the children under study were conceived *after* the conflict was initiated. Thus, the study loses the baseline, or non-conflict, level of maternal characteristics and fertility rates in each district. The authors must rely on the assumption that family planning decisions and behaviors were the same within a district during the lulls in conflict (which is their control cohort) as they were before conflict was ever initiated. It is highly plausible that the fertility demographics and behaviors during truly non-conflict times are quite different than during the temporary and unsustainable moments of low conflict used to generate variation in the study.

In addition to family planning behavioral responses, migration patterns cannot be assessed using this data. While the authors suggest migration was relatively low in general during this period, by citing that 94% of the births were to women living continuously in the same municipality since September 2000, this does not account for all the women (and thus their children) who out-migrated from the survey area

due to this intense conflict. Moreover, if the 6% that reported having moved did so systematically in a way correlated with potential fetal health (for example, mothers with more means moved to safer areas), this could lead to biased point estimates of the impact of violence on birth outcomes.

The authors are able to help assuage some of these concerns by focusing on those families that had two children born during the 3-year sample window. By comparing siblings, any potential biases between families arising from time-invariant characteristics are no longer relevant, as all comparisons are within the family. This strategy greatly increases the identification strength of the study, but raises some additional concerns when using this data in this setting.

Since the data window is so short, the sample remaining when only using mothers that gave birth twice in a three-year period has the potential to not reflect the general population. Additionally, since the conception decision for almost all of these births was made after the conflict had begun, and thus the potential for violence exposure was largely anticipated, the selectivity of the sample of mothers choosing to have more than 1 child during this time is plausibly exaggerated. Finally, while sibling fixed effects is an effective strategy to control for time-invariant characteristics of families/mothers, when used in a setting in which the conflict is not an unexpected shock, the potential for time-varying heterogeneity within a family driving the timing and health of a birth is exacerbated. With these limitations in mind, this work is still the strongest analysis of this topic in the literature, and a jumping off point for this study.

A second paper that should be mentioned is a working (by Florencia Torche and Andres Villarreal that, like this study, is interested in the question of how the surge in crime in Mexico has affected the birth outcomes of the exposed (2012). To examine this issue, they utilize INEGI monthly homicide data at the municipality level, pairing it with the birth certificate data of all children born between January

2008 and December 2010. The results from Torche and Villarreal's analysis are quite surprising and strikingly different than what has previously been found in the literature.

Their estimates suggest that children in Mexico exposed to additional homicides during the first few months of gestation show *increased* birth weight and *lower* probability of being born low birth weight. They suggest that it is increased prenatal care behavior, spurred on by early gestation exposure to conflict, specifically amongst the urban, low socioeconomic status mothers, which is driving these counter-intuitive findings. The strength of this work is that it utilizes official birth certificate data and the large number of observations allows for robust subsample analysis. While the richness of this study's data is quite useful, the time period under study and the lack of longitudinal data with sibling linkages makes it very hard to interpret the counter-intuitive findings as causal.

The researchers, due to lack of birth weight information in the birth certificate data before 2008, are limited to studying a sample in which the majority of the births were conceived after the violent surge had already begun. As mentioned in relation to Mansour and Rees's work, this is potentially very problematic when considering the systematic behavioral responses families may make during a time of conflict. Selective migration is one potential reaction to increased violence (or threat of violence). When the authors take a look at the impact of homicides on out-migration of childbearing aged women using 2005 and 2010 population counts, they find that women with exactly 9 years of education are more likely to out-migrate during times of greater violence. Additionally, this sensitivity analysis is only able to account for migration between 2005 and 2010, while temporary migration during pregnancy and delivery, which may be more reactive to local violence, cannot be captured.

A second possible response to violence is changing fertility behavior. Unfor-

tunately, the authors are unable to rigorously analyze whether there is a fertility response to violence because the data is all post-conflict initiation and thus a true baseline level is not available for comparison. That said, using data from 2008 to 2010 they find a positive relationship between municipality level homicide rates and birth rates. Thus, it seems that theoretically as well as in practice, using data that does not allow for control of systematic migration or between family unobserved heterogeneity to analyze the impact of violence on birth outcomes in Mexico, particularly when data is only available for the post conflict initiation period, leads to a selected sample and potentially misleading results.

4.2 Data

Properly estimating the impact of violence on individual health outcomes faces numerous empirical obstacles. In order to take on these challenges, this study will pair the INEGI monthly homicide data at the municipal level with the fortuitously timed and rich Mexican Family Life Survey (MxFLS).

The INEGI data provides information on all official reports of intentional homicides. A helpful feature of the data is that the label of "homicide" is assigned to a death using the World Health Organization's guidelines (ICD-10, Instituto Nacional de Estadstica), which should shield the data from regional differences in the classification or rate of reporting of a deceased as a homicide victim. One potential concern with this data is that it contains information only on registered homicides. If rates of reporting are significantly lower for cartel related homicides this data may serve as a very poor proxy for local conflict levels. Previous research has addressed this issue by comparing the INEGI data to organized crime related homicide data collected by the President's Office and found that the INEGI data captures the same trends found in the more explicit OCG-related President's data (Velasquez 2013). The advantage to and reason for using the INEGI data throughout the rest of the

analysis is its longer collection period, 1990-2011. Using data that spans both the pre-conflict and conflict periods allows the temporal variation of homicide rates in Mexico to be combined with the panel nature of the MxFLS.

The MxFLS is an ongoing longitudinal data set that is representative of the Mexican population in 2002. During the 2002 baseline survey, MxFLS1, information was collected on approximately 8,440 households and 35,600 individuals among 150 communities and 16 states throughout Mexico. The second wave, MxFLS2, was conducted in 2005-2006 and the third wave, MxFLS3, was started in 2009 and is currently in the final stages of fieldwork. The MxFLS was designed to follow all individuals in baseline households (and children born to these individuals since baseline) and has had great success in keeping low levels of attrition; with over 89% of the panel respondents being re-interviewed in MxFLS2 and similar re-contact rates are anticipated for MxFLS3.

More than just pointing to the low attrition rate in this survey though, it is of first order importance to test whether the sample of interest (childbearing aged women) are more likely to attrite from the sample due to a rise in violence in their municipality of residence. In order to formally test whether attrition amongst our population of interest is being driven by potential violence exposure, an analysis is conducted on women aged 7-42 in MxFLS1 (thus approximately 14-49 years old and eligible for the birth history component by MxFLS3) that estimates the relationship between the change in the homicide rate from 2002 to 2009 in the respondent's MxFLS1 municipality of residence and their likelihood of attriting from the MxFLS. Similarly, an analysis using women aged 10-45 in MxFLS2 examines if the change in violence between 2005 and 2009 in the respondent's MxFLS2 municipality predicted their future attrition. In addition to simply seeing if attrition is related to pending increases in local violence in general, specifications are also conducted in which the homicide rate variable is interacted with MxFLS1 or MxFLS2 characteristics of the

respondent to determine if attrition amongst subgroups of the population is predicted by increased conflict.

Examination of this issue reveals that attrition was quite low amongst this group of respondents: 13.4% between baseline and the third wave and less than 9% between the second and third wave (Table 4.1). Furthermore, no statistically significant relationship is detected in each analysis; suggesting that potential exposure to conflict was not a determining factor of attrition from the MxFLS3 sample by childbearing age women.

One particularly valuable aspect of the MxFLS, for the purposes of this study, is the fact that the timing of the survey waves provides a useful snap shot of Mexico before and during the major rise in conflict. The first follow-up was conducted between 2005 and 2006, a period of low levels of violence, and the second follow-up was performed from 2009 to 2013, during times of extremely elevated violence.

Figures 4.3-4.6 show the municipality homicide rate per 10,000 inhabitants for 2002, 2005, 2007, and 2009. Figures 4.3-4.5 provide a view of the conflict landscape during MxFLS1, MxFLS2, and an intermediate year between MxFLS2 and MxFLS3. The picture painted by these three maps is one of heterogeneous rates of violence, with homicides mainly concentrated across the United States border, a main drug trade route running along the Sinaloa-Durango and Sonora-Chihuahua borders up to the U.S.-Mexico border, and in the southern coasts of Michoacn and Guerrero. By 2009 though, as seen in Figure 4.6, the image of violence in Mexico was much different. Figure 4.6 shows that the conflict had intensified and spread across Mexico, and areas like the interior of Durango and southern Sonora, which previously were off the main drug trade routes and thus shielded from most of the violence, were now at the center of the drug war.

While the magnitude of the conflict has risen significantly in the last few years across Mexico, the level of the change across municipalities varies a great deal. For

example, between 2005 and 2009 the range of growth rates in homicides between municipalities spanned from a dreadful 30-fold increase in one area to an 80% decrease in another. Thus, along with the temporal variation in violence, this analysis will also be able to exploit the geographic distribution of conflict exposure across municipalities. Given that we have a great deal of variation in conflict intensity growth between municipalities, an open question is whether this violence heterogeneity actually reflects underlying trends in other municipality characteristics. If this were the case, it would raise concerns that an analysis of the impact of violence on birth outcomes would actually be picking up the effect of some other municipality trend on in-utero health. While it seems unlikely, due to the suddenness and well-documented origins of the change in the conflict environment, that some other municipality characteristic trend would be generating the temporal and geographic heterogeneity in violence seen in Mexico, we will formally explore this concern.

To examine this question a rich set of pre-escalation of violence trends of the 135 baseline MxFLS municipalities are used to predict each municipality's 2009 homicide rate, as well as the change in homicide rate in each municipality between 2005 and 2009. Trends were created using the IPUMS samples of the 2000 and 2005 Mexican censuses and the MxFLS1 and MxFLS2 survey waves.[4] Table 4.2 displays the results of these analyses. In both specifications, the estimates strongly suggest that

[4] From the Census the municipality trend between 2000 and 2005 are included for, the share of households with basic utilities (piped water, sewage system, electricity, television), the share of working age adults (21-65 years old) with particular levels of education ("less than primary" and "at least high school grad"), the ability to speak the indigenous language, and literacy, as well as, the overall share of different age groups (<18 and 18 to 65) and average educational attainment. From the MxFLS the municipality trend between MxFLS1 and MxFLS2 are included for, the share of adults (older than 18) that are married, are employed (by gender), are self-employed (by gender), live in a rural area, have a relative in the U.S., have thoughts of future migration, have fear during the day, have fear in the night, as well as, the average household size, hourly earnings (by gender), and per capita household expenditure. Also from the MxFLS municipality trends between MxFLS1 and MxFLS2 are included for the average report of increased domestic violence, the presence of vandalism, the level of police presence, the number of schools at various education levels (primary, junior high, and high school), and the rate of "poor" households.

pre-conflict trends in municipality characteristics were unrelated to future homicide rates.[5]

An additional advantage of the MxFLS is the comprehensive set of variables it collects about its participants, including information about the individual's economic, social, and health outcomes and behavioral histories (migration, fertility, marriage). Furthermore, information of household expenditure and asset ownership is gathered as well. Moreover, by using the panel nature of the data, the very serious potential biases from selective and endogenous fertility and migratory patterns can be examined.

Most importantly for this project, though, are the detailed reproductive histories collected in the MxFLS. For example, in MxFLS3, all household member women between 14 and 49 are asked to provide information such as date of birth, birth weight, prenatal care behavior, and place and locality of delivery on all pregnancies that have occurred since the MxFLS2 interview.

Focusing on panel member women in order to maintain representativeness provides a sample of 1,850 live births since MxFLS2 to 1,608 women (Table 4.3, Column 1).[6] As mentioned in the previous section, properly identifying the impact of conflict on birth outcomes will be greatly improved by the use of sibling comparisons, for which there are 471 sibling births to 229 mothers (Table 4.3, Column 2).

Two potential concerns exist, though, when considering the use of these samples as the main populations of examination. First, since some of the interviews in MxFLS3 took place in late 2009 and beyond, some of these pregnancies were conceived after the surge in violence could no longer be thought of as a shock or

[5] While there is 1 coefficient in Column 1 and 3 coefficients in Column 2 significant at the 10% level, given the number of dependent variables (64), this is even fewer significant estimates than what would be expected by chance. Moreover, joint tests for each regression are non-significant.

[6] There are 2,087 live births since MxFLS2. To create the analysis sample, 128, were removed due to lack of birth date information. Additionally, 84 births were dropped due to missing birth weight data. Lastly, 20 twins and 5 duplicates were not included in the sample.

unanticipated. With the conflict intensity no longer being exogenous to family planning behavior, the children conceived during this time might be part of families that are significantly different in observed and unobserved ways that are also correlated with fetal health. While the use of sibling comparisons will help alleviate this issue to some extent, time-varying family characteristics that may lead a mother to have an additional child during a predictably high homicide period still present an issue for identification.

To combat this problem, analysis can be conducted on a sample that only includes births before July 1, 2009 (labeled as born before 3Q2009, Table 4.3, Columns 3 and 4). Using this sample, all of the births will have been conceived, approximately, at latest in the third quarter of 2008, eliminating around one-sixth of the births from the full sample and one-third of the births from the sibling sample. The reasoning for the timing of this sample selection is that, at this point, homicide rates had been elevated for only a few months, which generate variation in a key period of gestation, first trimester, but the violence was still relatively new and less predictable. In order to make sure issues of selective fertility based on anticipated violence does not continue to drive the results even after making this sampling choice, analyses limiting the observations further, to those born before the second quarter of 2009 or those born before 2009, are also conducted.

The second issue that exists for both of the sibling samples mentioned previously is that they rely on mothers that gave birth to multiple children in a 3-4 year period. This is problematic for two reasons. Firstly, this restriction leads to a fairly small number of observations, which serves to reduce the power of the analysis, constrain the number of additional controls that can be used, and limit the number of stratifications that can be run in order to parse out any heterogeneity in the estimated effect. Secondly, by only being able to include mothers that gave birth to multiple children in a relatively short time span, the sibling sample mothers end up having sig-

nificantly different characteristics than those in the full sample. For example, when comparing baseline characteristics of the mothers with only one child in column 3 with those with multiple births in column 4 it is apparent the sibling sample is significantly different in a few ways. Single-birth mothers are statistically significantly older and more likely to be married in 2002, and though not statistically significantly so, they are also earning more per month and living in households with higher per capita expenditure.[7] As such, the sibling sample may be drawn from lower on the socioeconomic status continuum.

To make the sibling and full sample more comparable, any birth since baseline (2002) to a mother in the MxFLS3 birth history is included, while continuing to exclude births after the second quarter of 2009 (Table 4.3, Columns 5 and 6). This sampling choice adds 320 births to the full sample and triples the size of the sibling sample. Importantly, now the only observed characteristic that significantly differs between mothers in the full sample and mothers in the sibling sample is that mothers with multiple births have significantly more education at baseline (though this difference is eliminated once age at baseline is controlled). This final sample (Table 4.3, Columns 5 and 6) will be treated as the preferred population because, along with being more representative, the increased size allows the use of additional controls and for the heterogeneity of the impact of violence to be explored through stratification.[8]

Using the nationally representative MxFLS, combined with INEGI's monthly homicide data at the municipality level, this study will be able to take advantage of large and unanticipated variations in violence exposure across regions and time and pair them with a sibling fixed effect identification strategy.

[7] Conducting the same comparison of single birth mothers in Column 1 to multiple birth mothers in Column 2, finds that the single birth mothers are significantly older, more likely to be married, earn more, and live in higher per capita expenditure households at baseline.

[8] Analysis using the sample focused solely on the MxFLS3 birth history births before the third quarter of 2009 (Table 4.3, Column 3 and 4) provides qualitatively similar and quantitatively larger estimates of the effect found when using the preferred sample.

4.3 Empirical Strategy and Results

4.3.1 Behavioral Responses: Migration and Fertility

A violent conflict of the scale currently faced by Mexican citizens will almost surely result in systematic behaviors being taken by a selected group of the exposed in order to alleviate the potential negative spillovers to their well-being. Recognizing, analyzing, and accounting for these responses is imperative to any study of the Mexican drug war's impact on individual outcomes. Specifically, in the case of studying the effect of in-utero exposure to violence on fetal health, two behavioral responses must be addressed: migration and family planning/fertility.

Systematic migration as a result of a realized or impending surge in crime has the potential to change the composition of individuals exposed to violence and lead to biased results. For example, if mothers with a larger preference for safety are more likely to move away from high crime areas, and this safety preference also leads these mothers to take additional pro-health behaviors, the high crime areas would disproportionately be left with less healthy mothers and thus lower quality births without violence exposure being the cause. As such, it is important to determine whether migration decisions are being driven by potential exposure to violence.

In order to examine this issue, three measures of migration behavior will be analyzed. The first measure is simply an indicator for whether the interview municipality in MxFLS3 is different than the interview municipality in MxFLS2, which is the case for approximately 7% of the mothers (Table 4.4, Columns 1 and 2). The second identifier of migration is an indicator of whether the respondent has answered that they have moved from their MxFLS2 locality for longer than one year at any time between the MxFLS2 interview and the MxFLS3 interview, which represents approximately 16% of the women (Table 4.4, Columns 3 and 4). Finally, the last and most sensitive measure of migration is an indicator of whether the respondent answered

that they have moved from their MxFLS2 locality for longer than one month at any time between the MxFLS2 interview and the MxFLS3 interview, which accounts for about 17% of the mothers in each sample (Table 4.4, Columns 5 and 6).

Table 4.4 presents results of regressions on our sample of mothers that test whether the change in violence between 2005 and 2009 in the mother's municipality of residence in MxFLS2 was predictive of their decision to migrate. To estimate the relationship between migration and potential exposure as carefully as possible, and to avoid spurious correlations, each of these regressions additionally controls for various individual and household characteristics in MxFLS2 (age fixed effects, education, marital status, earnings, employment, rural status, household size, and household per capita expenditure), the municipality characteristic trends presented in Table 4.2, MxFLS2 state of residence fixed effects, as well as, year and month of MxFLS3 interview fixed effects.

$$m_{ij} = \alpha + \pi \Delta HOM_j + \gamma_{YOI} + \gamma_{MOI} + \sigma_{STATE} + \beta^{'} X_i + \psi^{'} \rho_j + \epsilon_{ij} \tag{4.1}$$

This specification is represented in equation (4.1), where m_{ij} is the migration decision of individual i, that resides at baseline in municipality j, ΔHOM_j captures the change in the homicide rate between 2005 and 2009 in municipality j, X_i is a vector of individual characteristics measured in MxFLS2, ρ_j is a vector of municipality characteristic trends, γ_{YOI} are indicators for the year of interview in MxFLS3, γ_{MOI} are indicators for the month of interview in MxFLS3, and σ_{STATE} are indicators for the state of residence in MxFLS2.

Moreover, it is also important to examine if the migration behavior of certain subgroups of the population was sensitive to local violent conflict. If potential violent crime exposure caused particular groups of mothers to migrate, and this systematic behavior was unaccounted for, it would create bias in the estimates of the impact of violence on birth outcomes. To explore if this is a concern, equation (4.1) is also

estimated with characteristics of the mother (age, age squared, education, marital status, earnings, employment, rural status, household size, and household per capita expenditure) measured in MxFLS2 interacted with the change in the local homicide rate.

The analyses of both versions of equation (4.1) are presented in Table 4.4. Column 1 provides estimates using the measure of movement typically used when analyzing migration in birth outcome studies: whether the mother resides in the same place as the previous wave (or in some studies some specified prior date). Examination of the relationship between this measure and potential future homicide exposure suggest that future violence does not predict migration. Moreover, even adding more detail to the analysis, by exploring if within certain subgroups future local conflict predicts being interviewed in a different municipality than in the previous wave (Column 2), there appears to be no relationship between violence and migration behavior.

Using a migration measure that is more inclusive and short-term in nature (any migration away from MxFLS2 locality for at least a year) and possibly more relevant for pregnant women, provides a different interpretation of the relationship between migration and conflict exposure. Column 3 of Table 4.4 provides evidence that future local violence increases migration behavior amongst the mothers in the sample, with a 1 in 10,000 rise in the homicide rate increasing the probability of migration by 1.5%. Moreover, Column 4 of Table 4.4 suggests that the influence of violence on migration was particularly strong amongst mothers from rural areas, mothers that earned more, and mothers living in more wealthy households.[9]

This analysis strongly implies that not measuring short-term migration may provide misleading conclusions about the relationship between local conflict and migratory behavior. Additionally, since migration does appear to be a behavioral response

[9] These results hold when using an even more liberal measure of migration, any movement for at least a month from the MxFLS2 locality, found in Columns 5 and 6.

to potential violence exposure and that the response is systematically taken by subgroups within the population of mothers, failing to control for migration in an analysis of the impact of violent crime on birth outcomes would produce non-trivial bias in the estimates.[10] Given this serious threat to identification, the issue of endogenous migration will be addressed directly when developing the methodology to test the effect of local homicides on birth outcomes in the next subsection.

Turning to fertility behavior, exposure to local violence has the potential to impact birth rates in the effected area in a few first order ways. First, certain families may actively try not to conceive a child during a time of intense conflict. They may see this environment as dangerous for the health of the mother, dangerous for the health of the child, or infeasible due to a loss in resources. If these families are drawn from a specific part of the birth outcome distribution, not accounting for this composition change will bias results. An additional possibility is that local birth rates are being driven by selective migration, with families more or less likely to conceive choosing to move away from local crime. Finally, birth rates may be impacted directly by violence exposure. If the anxiety and/or resource restrictions caused by local violence are severe enough, fetal health may deteriorate to a point that a non-marginal number of pregnancies may be lost.

To examine the impact of local violence on monthly birth rates of MxFLS1 municipalities the following regression was estimated:

[10] One additional note, even if the decisions to migrate was not impacted by potential violence, the destinations of those movements may be changed in a systematic way. If, rather than level of migration, the location of migratory flows changed in a way correlated with both anticipated future homicide exposure as well as fetal health, this would skew results, as well.

$$BR_{jym} = \sum_{i=10}^{15} \pi_i HOM(\text{i mos. before } ym)_{jym} + \pi_1 HOM(\text{9-7 mos. before } ym)_{jym}$$
$$+\pi_2 HOM(\text{6-4 mos. before } ym)_{jym} + \pi_3 HOM(\text{3-1 mos. before } ym)_{jym}$$
$$+\alpha + \gamma_y + \gamma_m + \delta_j + \sigma_{STATE,y} + \epsilon_{jym} \tag{4.2}$$

where, BR, is the birth rate of the relevant group in year y, month m, and municipality j, $\sum_{i=10}^{15} \pi_i HOM(\text{i mos. before } ym)_{jym}$, represents the municipality homicide rate for each month from 10 to 15 months before the birth rate in year y, month m, and municipality j, $HOM(\text{9-7 mos. before } ym)_{jym}$, $HOM(\text{6-4 mos. before } ym)_{jym}$, $HOM(\text{3-1 mos. before } ym)_{jym}$, represent the municipality homicide rates over an approximation of the first, second, and third trimester of the outcome birth rate, respectively, and γ_y, γ_m, δ_j, $\sigma_{STATE,y}$ represent fixed effects at the year, month, municipality, and state times year level, respectively.

The numerator for the outcome birth rate, BR, was calculated as the number of births in the MxFLS reproductive history. To create the denominator for the birth rate, the number of women 14 to 49 years old (and thus eligible to complete the reproductive history) in each wave of the MxFLS were counted and considered the base January population in the year following the initiation of that wave of the MxFLS. Thus, the count for MxFLS1 was used for January 2003, the count for MxFLS2 was used for January 2006, and the count for MxFLS3 was used for January 2010. Then a linear imputation method was used to fill in the months in between waves. The same strategy was used when constructing birth rates for women with less than 9 years of education and for women with at least 9 years of education (compulsory level). The time period of this analysis runs to June 2009, as this is the latest date for which the MxFLS3 reproductive history should contain all births up

to that point, as no MxFLS3 interview took place before June 2009.

Table 4.5 displays the findings from estimation of equation (4.2). The results from Column 1 suggest that local homicide rates before conception and all the way through pregnancy did not significantly change overall birth rates. Additionally, when looking at a lower education subgroup (<9 years of education), there does not appear to be a statistical relationship between violence just before or during gestation on birth rates (Table 4.5, Column 2). Interestingly though, for higher educated women ($\geqslant$9 years of education), even when controlling for a great deal of temporal, geographic, and even geographic time trend variation through the inclusion of fixed effects, it appears there is a response in birth rates to conflict.

The estimates in Column 3 of Table 4.5 suggest that educated women, when faced with increasing violence during the time leading up to a potential conception date, are less likely to reproduce. Moreover, increased homicide exposure in the area of the second trimester also seems to lower birth rates. This second finding may be caused by families being able to forecast conflict intensity and deciding not to have children when violence is expected to rise during the 4 to 6 months before birth, or perhaps, within the subgroup of higher educated women, homicide exposure in the second trimester has a non-trivial impact on being able to produce a live birth.

This finding serves to underline the importance of using a sibling fixed effects identification strategy in order to estimate the impact of local homicide rates on fetal health. While the current alternative in the field is to simply control for temporal and geographic heterogeneity through time and location fixed effects, the results in Table 4.5 suggest this strategy would contain biased estimates. In this specific case it appears a method of time and location fixed effects would not control for a compositional change amongst the second trimester exposed group of educated women and perhaps wrongly attribute a reduction in birth outcomes to violence exposure.

4.3.2 Birth Outcomes: General Results

This section will present results of an evaluation of the impact of local homicide rates during gestation, constructed as 1 to 9 months before birth, on birth outcomes. This analysis is an extension and contribution to previous work on conflict and in-utero health as it utilizes a sudden increase in violence, data that spans the non-conflict and conflict periods, and a longitudinal survey, which helps to address some of the potential concerns raised by systematic behavioral responses by different types of mothers.

Issues related to each of the two behavioral responses discussed in Subsection 4.3.1 inform and guide the identification strategy used to estimate the impact of local homicide rates on birth outcomes. As mentioned previously, migration decisions for mothers in the sample were significantly influenced by exposure to conflict. In order to take on this issue of systematic behavioral response directly, the identification strategy employed in this analysis takes an intent-to-treat approach. Specifically, exposure intensity during gestation for each birth will be calculated as the homicide rate during that time period in the mother's baseline (MxFLS1) municipality of residence, rather than the municipality of residence at birth. While this approach may attenuate the estimate of the impact of local violence on birth outcomes, it alleviates concerns that endogenous migration behavior is driving the results.

Also, as mentioned in Subsection 4.3.1, there appears to be a reduction in birth rates amongst more educated women when they experience higher conflict intensity prior to conception and around the second trimester, even after controlling for regional and temporal fixed effects. This bias causing systematic behavior is an example of the composition issues that may exist when only temporal and geographic heterogeneity is controlled. Moreover, as with any survey, the amount of covariates available to use as controls between mothers is limited. This limitation may lead

to the misidentification of heterogeneity in health related preferences and behaviors of mothers differentially exposed to local homicide rates as an effect of violence on birth outcomes.

These issues of composition change and omitted variable bias are ever-present in studies of fetal health. The strategy that will be used in this study to address these concerns is the use of sibling comparisons. By only making comparisons within a family, time invariant characteristics or preferences of the mother/household are controlled. Additionally, in an attempt to limit the potential for time-varying within-family behavioral changes related to violence exposure biasing the results, samples that are limited to births conceived before violence levels could be predictably anticipated are utilized. Finally, available time-varying characteristics (mother's education, age at birth, employment status, earnings per month, and marital status, as well as, household size, rural status and per capita expenditure) between baseline and MxFLS2 are included. Since these characteristics are not measured at the time of birth (except for age at birth) they are assigned as follows (except for age at birth): if a birth is from the MxFLS3 reproductive history they are assigned the time-varying characteristic reported in MxFLS2 and if the birth is from the MxFLS2 reproductive history they are assigned the time-varying characteristic reported in MxFLS1.

Standard temporal (month of birth, year of birth, month of interview, and year of interview) and geographic (municipality of birth) fixed effects are also included in some specifications to control for any spurious relationship between the date of interview, as well as, the time and place of the birth and the birth outcome, which is unrelated to violence exposure. Furthermore, when sample sizes are large enough, state of birth interacted with year of birth fixed effects are included to remove additional state-year trend unobserved heterogeneity from the analysis.

Finally, local homicide rates from before conception and after birth will be in-

cluded as controls. The results from Subsection 4.3.1 make it clear that behaviors related to family planning are being impacted by local violence several months before the potential conception month. To account for these fertility behaviors as well as other behaviors related to violence that may change the composition of maternal characteristics, even in a within-family comparison, the local homicide rates for the 6 months before conception, 15 to 10 months before birth, are included. In addition, it may be the case that local violence in the time surrounding a birth has a relationship to birth outcomes that is unrelated to its impact on the fetal health of the child. As such, the homicide rate for the 6 months after birth are additionally added to the regression, as these homicides should be related to the local homicide rate during pregnancy but unrelated to birth outcomes and serve to control for any additional spurious relationship.

The empirical strategy can be generalized in the following regression framework:

$$\begin{aligned} b_{ijtmk} &= \pi_1 HOM(\text{9-7 mos. before birth})_{tk} + \pi_2 HOM(\text{6-4 mos. before birth})_{tk} \\ &+\pi_3 HOM(\text{3-1 mos. before birth})_{tk} + \psi' X_{itm} + \gamma_{YOB} + \gamma_{MOB} + \eta_{YOI} + \eta_{MOI} + \delta_j \\ &+\sigma_{STATE,y} + \sum_{i=10}^{15} \pi_i HOM(\text{i mos. before birth})_{tk} + \sum_{i=1}^{6} \pi_{-i} HOM(\text{i mos. after birth})_{tk} \\ &+\upsilon_m + \epsilon_{ijtmk} \end{aligned} \tag{4.3}$$

where b is the birth outcome of individual i, born in municipality j, in time t, to mother m, that resided in municipality k at baseline, υ_m captures sibling fixed effects,γ_{YOB} are indicators of the year of birth, γ_{YOB} are indicators of the month of birth, η_{YOI} are indicators of the year of interview, η_{MOI} are indicators of the month of interview, δ_j are municipality of birth fixed effects, $\sigma_{STATE,y}$ are state of birth interacted with year of birth fixed effects, X_{itm} is a vector of individual (gender, birth order fixed effects, and survey wave) and time-varying mother/household characteristics, and $HOM(\text{i mos. before birth})_{tk}$, $HOM(\text{9-7 mos. before birth})_{tk}$,

HOM(6-4 mos. before birth)$_{tk}$, HOM(3-1 mos. before birth)$_{tk}$, and HOM(i mos. after birth)$_{tk}$ are homicide rates in the municipality of mother's baseline residence, k, during specific periods before, during, and after gestation of individual, i.

Table 4.6 presents the estimates from specifications that build up to equation (4.3). In Column 1, using a specification without sibling fixed effects, it appears that local violence in the middle of gestation, 4 to 6 months before birth, is negatively related to a loss in birth weight, while exposure during the rest of gestation is non-significant. This estimate though may be driven by the change in fertility behavior presented in Table 4.5. Table 4.5 suggested that the comparison group of mothers exposed to violence in the second trimester is composed of too few births to women with at least compulsory education, which would lead to downward biased estimates. Even after adding the full set of controls other than the sibling fixed effects (Column 2) and moving to the sibling sample (Column 3), the magnitude of the estimate on exposure 4 to 6 months before birth is essentially unaffected and exposure in all other parts of gestation continue to have no relationship to birth outcomes.

In Column 4 the sibling fixed effects are introduced and have a dramatic effect on the results. First of all, once maternal fixed effects are included, the sign of the effect from exposure 4 to 6 months before birth is reversed and the absolute magnitude is greatly diminished. Additionally, the estimates now suggest that experience of increased local violence 7 to 9 months before birth leads to statistically significantly reduced birth weight. This non-trivial change in the overall set of results points to the misleading conclusions that can be drawn when an analysis of crime on birth outcomes fails to control for unobserved heterogeneity between mothers/families. Moreover, as additional controls are added (Columns 5-7) the magnitude of the early gestation effect only grows.

To give some guidance to interpreting the results, the average homicide rate in

Mexico between the pre-escalation of violence period of 2005-2007 and 2009 rose by approximately 1 homicide per 10,000 in MxFLS1 municipalities, which would produce a rise of around .25 homicides per 10,000 in the 3 month homicide rate. Thus, the results in Table 4.6 estimate that the loss in birth weight resulting from the average 3-month increase in violence in Mexico between the pre-conflict period and 2009 is 75 grams (300*.25).[11]

An alternative way to conceptualize the estimates is to calculate the impact on birth weight of 1 additional homicide in a representative municipality. The median 2009 population amongst MxFLS1 municipalities is around 60,000 people, thus according to estimates in Table 4.6, one extra homicide during early gestation in a municipality of this size would generate a 50 gram decrease in birth weight amongst the exposed.[12]

While Table 4.6 restricts births to those born before July 2009 in order to shield the estimates from births in which local violence was easily predictable by the families, it is important to check that this restriction has gone far enough. To test the robustness of the initial findings, Table 4.7 contains the results of the full specification from equation (4.3) using samples limited to children born before April 2009 and before January 2009, respectively. Columns 2 and 3 of Table 4.7 display these findings. These estimates suggest that the inclusion of children born between January and June 2009 is not driving the main results, as these more restrictive samples produce very similar results.

Additionally, since all the birth weight estimates are based on self-reports, it may be the case that the birth weight measures of children born outside a medical facility contain a great deal of error that is correlated with local violence exposure.

[11] Moreover, 2009 was just the beginning of the Mexican drug war surge in violence. By 2011, the annual rate had risen by around an additional .5 homicides per 10,000.

[12] Calculated as (-300*10,000)/60,000.

Thus, an additional analysis was conducted on only the subsample of children born in a hospital or clinic (Table 4.7, Column 4). While the level of significance on the impact of early gestation exposure to violent crime is reduced, due to the increased size of the standard errors, the point estimate remains marginally statistically significant and the magnitude of the estimate is even larger (>15%) than the result in Table 4.6.[13] In summary, the analysis of the impact of increased local conflict on birth weight consistently confirms that early gestation exposure leads to significantly poorer outcomes.

To provide even more information about how local conflict is impacting birth outcomes, analysis on the probability of a birth falling into one of the commonly used categories of poor fetal health, low birth weight (<2,500g) is conducted and presented in Table 4.8. Column 1 provides the estimates of the probability of being less than 2,500 grams at birth and, due to stacking at 2,500 grams in the data, Column 2 presents the results when those listed as weighing exactly 2,500 grams are included as low birth weight as well. Both estimates are in concordance with the previous findings as they suggest that exposure to greater local violence in the first few months of gestation lead to an increase in the probability of falling into the category of low birth weight.

Using a 3-month shock in the early gestation homicide rate of .25/10,000 as a base, these results suggest that the probability of being designated as low birth weight rises by 3-5%, which relative to a base of 7-10% represents a 40-50% increase in this risk factor.[14]

[13] As an additional way to provide evidence of whether measurement error/recall bias in the reported birth weights is correlated with violence exposure, equation (4.3) was also estimated using a measure of stacking as the dependent variable. Stacking was measured as having a birth weight that ended in .0 or .5 kilograms. Conducting this analysis provides no evidence that stacking in birth weights is related to violence exposure.

[14] Alternatively, the impact of one additional homicide, during early gestation, in a municipality of 60,000 people is a 2-4% increase in the probability of being born low birth weight. Which is an effect size of 25-33% when scaled against the mean low birth rate incidence.

Another important aspect of a birth is its gestational length. Unfortunately the question regarding the number of weeks each pregnancy lasted was not asked to all mothers. The only available information about gestational length for all births comes from the following question asked to mothers: "Was your child from pregnancy #x born premature?" The rate of answering this question "Yes" was quite high, 16.5% for the sample under examination, while the expected rate of prematurity in Mexico is closer to 7-10%. The estimates when using this measure as the dependent variable in (4.3) are found in Column 3 of Table 4.8. Surprisingly there is a predicted reduction of self-assigned premature births with increased local homicide rates in mid pregnancy, as well as, a potential increased risk if exposure is late in pregnancy. Given the lack of clarity in what the premature birth self-assignment measure is capturing, though, it is quite difficult to interpret this result.

Lastly, the MxFLS asks the mothers various details of the delivery of each child, including a question of whether there were any complications or difficulties with each pregnancy. Column 4 of Table 4.8 provides the results of estimating (4.3) using this measure as the dependent variable. Evaluating this analysis there does not appear to be any relationship between gestational violence exposure and pregnancy complications.

In summary, children born to mothers that were exposed in early gestation to the recent surge in conflict caused by the Mexican drug war have substantially and statistically significantly poorer birth outcomes. Moreover, the general findings are consistently replicated over several samples and multiple layers of additional controls.

4.3.3 Birth Outcomes: Effect Heterogeneity

This section will be focused on exploring whether the effect of local violence on birth outcomes is significantly larger for families/mothers of lower socioeconomic status. There are several reasons to think this group would be particularly susceptible to

the impact of violence on birth outcomes. First of all, there is research that suggests that the most disadvantaged groups are the most likely targets of cartel victimization (Diaz-Cayeros et al., 2011). Moreover, due to this group's limited means, they may be less able to compensate for negative health shocks. Specifically, the analysis that is conducted in this study is an examination of the impact of local violence on families in the bottom 50% of the per capita expenditure distribution at baseline or births to mothers with less than 9 years of education (compulsory level), respectively (Table 4.9).[15]

For this analysis, mother's education was assessed in two ways: educational attainment at baseline and educational attainment in MxFLS3. The advantage to using baseline education is that it is insulated from the potential endogenous effect of local violence exposure. On the other hand, by using education in MxFLS3 the measure will reflect completed education for the younger mothers in the sample. Results are similar when using either measure.

Columns 2-4 in Table 4.9 provide clear evidence that the birth weights of children born to families/mothers with lower socioeconomic status are impacted substantially harder by exposure to local violence early in gestation. In both cases the magnitude of the coefficient is more than double the size of the equivalent estimate (found in Column 1). Using .25/10,000 as a base for a 3-month early gestation impact of the Mexican drug war shock, this translates to a reduction in birth weight of around 120-125 grams.[16]

[15] Due to the reduced sample size, state of birth interacted with year of birth fixed effects are omitted.

[16] Alternatively, this estimate suggests an 80-84 gram drop in birth weight due to early gestation exposure to one additional homicide in a municipality with a population of 60,000 for lower socioeconomic status mothers.

4.3.4 Prenatal Care

An additional analysis that can be explored in this data is the impact local violence exposure had on the number and timing of a mother's prenatal care visits during each pregnancy. Tables 4.10 and 4.11 present the results of using the number of prenatal care visits and the probability of initiating prenatal care in the first two months of pregnancy as the dependent variable in equation (4.3) for the entire population, as well as, the three subgroups used in the previous subsection, respectively.

In Column 1 of Table 4.10 the estimates of equation (4.3) suggest that violence exposure early in gestation led to significantly fewer overall prenatal care visits. The heterogeneity of this result, though, is less clear, as those born to families in the bottom 50 percent of the per capita expenditure distribution in MxFLS1 or born to mothers with less than 9 years of education in MxFLS3 (Columns 2 and 4) have an exacerbated effect of early gestation local violence on prenatal care visits, while mothers with less than compulsory education in MxFLS1 (Column 3) face qualitatively the same impact. Interestingly, there appears to be some evidence that elevated exposure to increased local homicide rates late in pregnancy encourage additional uptake of prenatal care amongst the low socioeconomic status subgroups.

The sheer number of prenatal care visits, though, is only one component of prenatal care utilization, as several studies have shown that birth outcomes are significantly improved when prenatal care is initiated earlier in pregnancy (Rosenzweig and Schultz, 1983; Jewell and Triunfo, 2006; Jewell, 2007; Wehby et al., 2009). Table 4.11 contains the results of estimation of equation (4.3) when using the probability of prenatal care initiation in the first two months as the dependent variable.

As in Table 4.10, we see that exposure to local violence early in pregnancy led to poorer prenatal care, with mothers statistically significantly delaying prenatal care initiation, though this effect is not consistently stronger for low socioeconomic status

mothers. Also there is more evidence that the forward looking expectation of greater violence exposure late in pregnancy led to pro-health behaviors early in pregnancy amongst lower socioeconomic status families.

While these results display a very interesting behavioral response to local violence exposure, they do not seem to be the primary mechanism driving the poorer birth outcomes of exposed mothers, as the inclusion of both prenatal care measures as independent variables in equation (4.3) only partially (5-22%) attenuates the results from Tables 4.6 and 4.8. Specifically, including the number of prenatal care visits and whether prenatal care initiation began in the first two moths of pregnancy into equation (4.3) reduces the estimate of the effect of increased conflict exposure 9 to 7 months before birth from 299.9 to 281.1, 10.7% to 8.3%, and 21.2% to 20.1% for birth weight, being born <2,500 grams, and being born ⩽2,500 grams, respectively.

4.4 Discussion

4.4.1 Relative Size of the Effect

The analysis presented in this study makes a strong and consistent statement that exposure to local violence in early pregnancy leads to statistically significantly smaller births. Placing the size of the effect in context, though, is of first order importance, as it provides guidance for determining the severity of this concealed cost of crime and conflict.

One place to start when assessing the real toll on society caused by violence on birth outcomes is neonatal mortality.[17] Neonatal mortality has been shown to have a strong relationship to birth size. Almond, Chay, and Lee's estimate that a 100 gram increase in birth weight leads to a reduction of 1.5 neonatal deaths per 1,000 births (2005). Applying this to the findings presented previously suggests that exposure to a .25 per 10,000 increase in the homicide rate in early gestation results in more than

[17] Neonatal mortality is typically defined as the death of a live birth within 28 days.

1 additional neonatal death per 1,000 births, or an increase of almost 2 deaths per 1,000 births amongst mothers of lower socioeconomic status.[18]

A different way to provide perspective on the magnitude of the adverse impact of the Mexican drug war on fetal health is to compare its effect to commonly cited drivers of birth outcomes such as nutrition and maternal smoking. The Supplemental Nutrition Program for Women, Infants, and Children (WIC) and the Food Stamp Program (FSP) in the United States have been evaluated in terms of their positive effect on birth weight by Hoynes et al. and Almond et al., respectively (2011).

Hoynes et al. estimate a 2 gram effect of WIC on the average population and a 18-29 gram impact amongst participants, and Almond et al. find that FSP led to birth weight increases of 2-5 grams in general and 15-40 grams amongst the treated (2011). These effect sizes suggest that the impact of these programs, even amongst the highly selected participants, is considerably smaller than the harmful influence of being in utero during high levels of local violence.

Smoking, the most commonly excepted environmental risk factor of fetal health, provides an additional way to contextualize the size of the impact of the Mexican drug war. Taking the estimates produced from multiple strategies to assess the effect of maternal smoking on birth weight provides a general finding of around a 200-230 gram and a 100-130 gram loss in birth weight for children born to women that were heavy (11+ cigarettes a day) and light smokers during pregnancy, respectively (Rosenzweig and Shultz, 1983; Sexton and Hebel, 1984; Brooke et al., 1989; Wilcox, 1993; Almond et al., 2005; Ward et al., 2007). This suggests that the impact of the rise in violence in Mexico between the pre-conflict period of 2005-2007 and 2009 on birth weight was at least one-third and two-thirds the size of the effect of having a

[18] Additionally, using the estimates in Almond, Chay, and Lee as a guide, the results presented in this study suggest exposure in early gestation to a conflict shock of the size of the shift between the pre-escalation of violence period of 2005-2007 and 2009 in Mexico leads to more than a 1.5 and 2.5 increase in deaths per 1,000 births in the first year of life for all and lower socioeconomic mothers, respectively (2005).

heavy and light smoking mother during gestation, respectively. If focusing on the larger harm done to the birth outcomes of the mothers of lower socioeconomic status, the violence in Mexico had an effect half the size of having a heavy smoking mother or was equivalent to being exposed to a lightly smoking mother during gestation.

A final way to evaluate the magnitude of the effect of the Mexican drug war on fetal health is to compare its adverse impact to the gains achieved in Mexico by *Oportunidades/PROGRESA*, a government social assistance program partially designed to improve birth outcomes of participating women. *Oportunidades* (formerly *PROGRESA*) is a large-scale conditional cash transfer (CCT) program in Mexico that targets poorer families and ties compensation to investment in the education and health of the household's children. One component of the program was a condition that pregnant women needed to complete a prearranged prenatal care plan, acquire specific nutritional supplements, and attend meetings that focus on pregnancy health education (Barber and Gertler 2008). Evaluation of the impact this program had on the birth outcomes of participating mothers suggests that the children exposed to *Oportunidades* in utero were born 127 grams heavier (Barber and Gertler 2008). This estimate mirrors the magnitude of the negative impact on birth weight of exposure to the Mexican drug war in early gestation for lower socioeconomic status mothers (the group most similar to the *Oportunidades* sample), suggesting this recent conflict could be eliminating the gains of one of the oldest and largest CCTs in existence.

4.4.2 Mechanisms

One area in which this analysis is unable to make particularly definitive statements is in terms of the relative importance of each of the potential pathways through which local violence impacts birth outcomes. The main avenues suggested by the literature for an effect of local violence on birth outcomes are: biological reaction to anxiety, poorer health behaviors (e.g. smoking, less exercise), decreased use of health care, or

constrained nutrient intake. Given the results mentioned in Subsection 4.3.4, while it appears prenatal care was reduced and delayed due to exposure to violence, this is not the primary cause of the reduction in birth weight, accounting for only around 10% of the effect.

Another mechanism worth considering is reduced nutrition. There is considerable evidence that increased local homicide rates led to poorer economic outcomes (employment, earnings, hourly wages) for the exposed adults (Dell, 2011; Velasquez, 2013; Robles et al., 2013). If this shock to economic outcomes served to restrict the budget constraint of the household, leading to less nutrients being consumed by a pregnant family member, this may directly impact the fetal health of the in utero child. Almond and Mazumder find that a fetus, provided limited nutrients due to the mother's experience of Ramadan, is statistically significantly reduced in size at birth (2011). This effect, though, is not restricted to early gestation exposure. Reduced nutrient intake due to Ramadan reduced birth weight amongst children exposed in the first trimester (20 grams), as well as, the second trimester (26 grams). The wider temporal scope of the effect of restricted nutrient intake, as compared to the limited timing of the effect of local homicides, suggests that this may not be the primary pathway through which violence is impacting birth outcomes, although it does not completely rule out its contribution. For instance, it may be the case that when a family experiences a shock in earnings in mid gestation, they reallocate resources specifically to avoid the pregnant mother facing reduced nutrition, whereas this is not the case when a family is faced with financial deficits early in a women's pregnancy.

A more compelling argument that nutrition is not the main driver of the results is that the effect size estimated by Almond and Mazumder, as well as, the various studies of nutrition programs mentioned previously, such as WIC (2-5 grams) and FSP (2-5 grams), is significantly smaller than those found in this study. Given that those studies focused directly on nutrition and found much smaller effects, it is less

likely that the hypothesized potential nutrient restriction from Mexican drug war exposure would be the leading mechanism resulting in the large negative estimates found in this study. Unfortunately, without detailed individual-level consumption data over the gestation period, the level, timing, and impact of potential nutrient restrictions cannot be assessed.

The mechanism most commonly thought to be operating on birth outcomes during conflict is the fetus's exposure to maternal anxiety. The timing of the effect estimated in this study, early gestation, matches up with the findings from the medical and economics literature on the impact of maternal mental distress on fetal health (Schulte et al., 1990; de Weerth and Buitelaar, 2005; Camacho, 2008; Torche, 2011; Brown, 2013). Moreover, while the results in this study are much larger than those found in Camacho or Brown (9 and 15 grams, respectively), they are of the magnitude of those reported by Torche (51 grams).[19] This suggests that if maternal stress is the first order pathway being exploited in this study, the violence and victimization caused by the Mexican drug war may be leveling the same amount of anxiety on its population as a natural disaster.

The final potential avenue that may be triggering the large influence local violence is having on birth outcomes is changing maternal health behaviors. If exposure to local conflict spurs mothers to engage in risk-taking and/or health-reducing behaviors such as smoking or reduced exercise, this would have a non-trivial impact on the health of the fetus. While the MxFLS does not have data on tobacco consumption

[19] One potential reason for the large disparity in the results between this study and the works by Camacho and Brown is that they may have been using stress shocks of a smaller magnitude. For instance, the exogenous stressor used in Camacho's work, landmine explosions, is arguably so random that it may not be a particularly strong signal of conflict intensity in that area at that moment and thus the 9 gram estimate is a reflection of anxiety only due to the recent detonations but not to any fear of personal harm or victimization. In the case of Brown's examination of the impact of the 9/11 terrorist attacks on the birth outcomes of exposed mothers, his estimate is attenuated as it must exclude mothers living in areas that were directly attacked (New York City and Washington D.C. metro area) in order to avoid confounding factors such as pollution and the subsequent reduction in economic activity.

or physical activity with timing information at the level of detail needed to test if these behaviors saw an uptick in expression during each pregnancy, analysis can be conducted to determine if exposure to local violence changes the smoking and exercise behavior of the mothers in this sample.

Utilizing the longitudinal nature of the MxFLS, a respondent's level of smoking or exercise when measured in MxFLS2 can be compared to the same respondents behavior in MxFLS3. Specifically, it is possible to determine if the amount of local violence experienced in the year leading up to the interview significantly changed health oriented behaviors while controlling for all time-invariant heterogeneity at the individual level. As before, in order to eliminate the issue of endogenous migration, homicide rate level is assigned based on the pre-escalation of violence location of residence, in this case the MxFLS2 municipality of residence.

To examine this relationship the following equation was estimated:

$$y_{itjk} = \alpha + v_i + \delta_j + \eta_{YOI} + \eta_{MOI} + \psi^{'} X_{itjk} + \pi HOM(\text{1-12 mos. before interview})_{tk} + \epsilon_{itjk} \quad (4.4)$$

where, y, is the health behavior of mother i, at time t, interviewed in municipality j, and residing in municipality, k, in MxFLS2, $HOM(\text{1-12 mos. before interview})_{tk}$, represents the homicide rate over the 12 months prior to the interview in the municipality the respondent resided in at MxFLS2 interview, v_i, η_{YOI}, η_{MOI}, and δ_j represent fixed effects at the individual, year of interview, month of interview, and municipality of interview level, and, X_{itjk} is a vector of time-varying individual (age, marital status, educational attainment, monthly earnings, and employment status) and household (household size, rural status, and per capita expenditure) characteristics.

Columns 1, 2, and 3 of Table 4.12 display the results of estimating equation (4.4) using the number of cigarettes smoked per week, the number of exercise days between

Monday and Friday, and the amount of exercise time per day as the dependent variable, respectively. The results provide evidence that these mothers were not spurred to change health behavior in a negative way due to local violence exposure. While these results indicate that the negative impact on birth outcomes due to local conflict exposure was not a result of increased smoking or decreased exercise, the analyses are not detailed enough to detect changes during pregnancy periods and can only be taken as suggestive evidence.

4.5 Conclusion

The sudden and horrific internal conflict in Mexico has cost the country thousands of lives and disseminated a widespread sense of insecurity amongst the non-combatants. Research has documented some of the explicit effects of the violence such as increased victimization (extortions, kidnappings) and losses of earnings and employment. What has been left unexplored is the toll the increased violence may have on the well-being of the next generation.

Medical and economic research has continually produced a link between birth outcomes and markers of long run health, education, and employment outcomes. This set of facts, paired with the potential mechanisms at play in Mexico that may effect fetal health (maternal stress, resource restriction, reduced prenatal care), provides reason to believe that the Mexican drug war can adversely impact the long-term trajectories of those exposed in utero.

Evaluating the effect of conflict in any region on individual-level health outcomes always faces the challenges of separating out spurious relationships, as well as, tracking and correcting for behavioral responses. With these concerns in mind, this analysis is conducted on a violent conflict that escalated swiftly and with a great deal of heterogeneity, using data that contains the non-conflict and conflict periods, allows for the documentation and control of the potential concerns raised by systematic

behavioral response, and can take advantage of sibling comparisons.

The estimates in this study suggest that the average level of exposure to the Mexican drug war causes damage to birth weight 10 times larger than the gains found in U.S federal nutrition programs and about one-half the size of being born to a mother that smokes during pregnancy. Moreover, for children of lower socioeconomic households, the adverse effect of the recent exposure is equivalent to the gains scene in the birth weight of pregnant mothers enrolled in the large and successful Mexican conditional cash transfer program, *Oportunidades*. Most disturbingly, the homicide rate in many municipalities in Mexico has only continued to rise since 2009, suggesting these effect sizes are in fact lower bounds of the overall toll this internal war has taken on the next generation of Mexicans citizens.

4.6 Tables and Figures

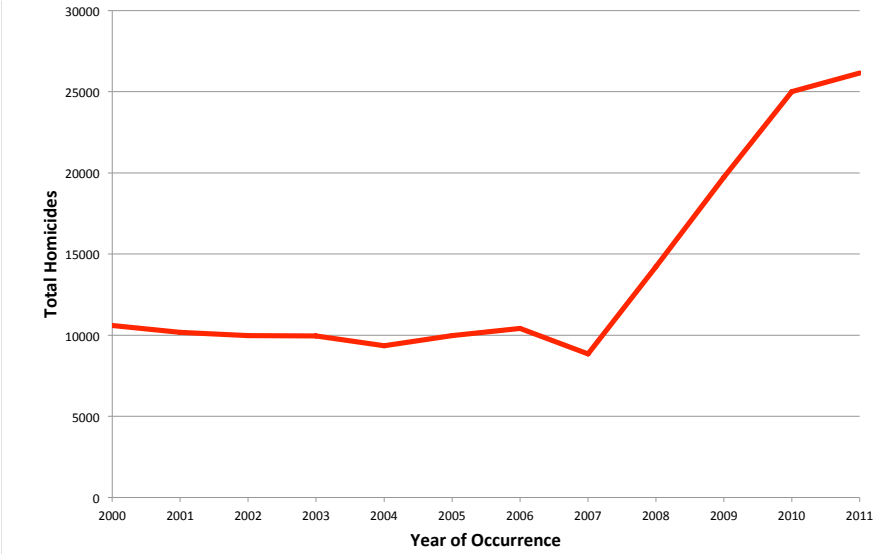


FIGURE 4.1: Total Homicides by Year in Mexico

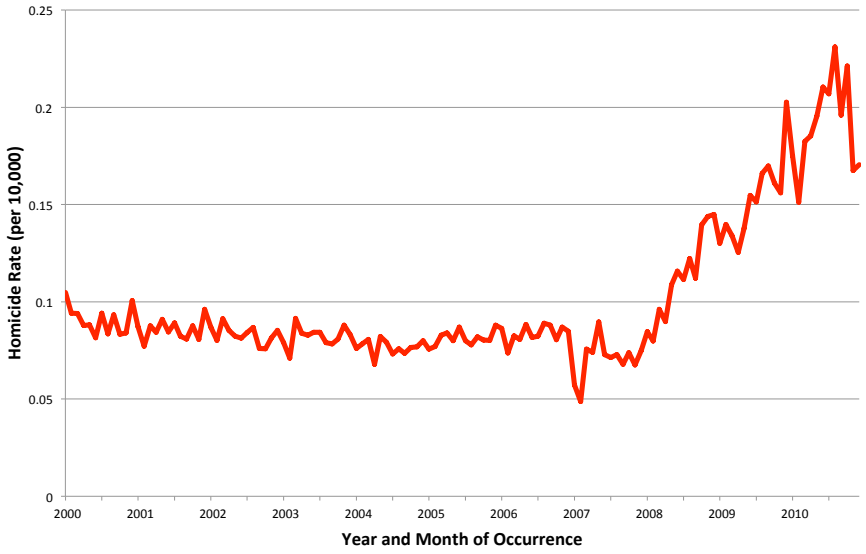


FIGURE 4.2: Homicide Rate by Year and Month in Mexico (per 10,000)

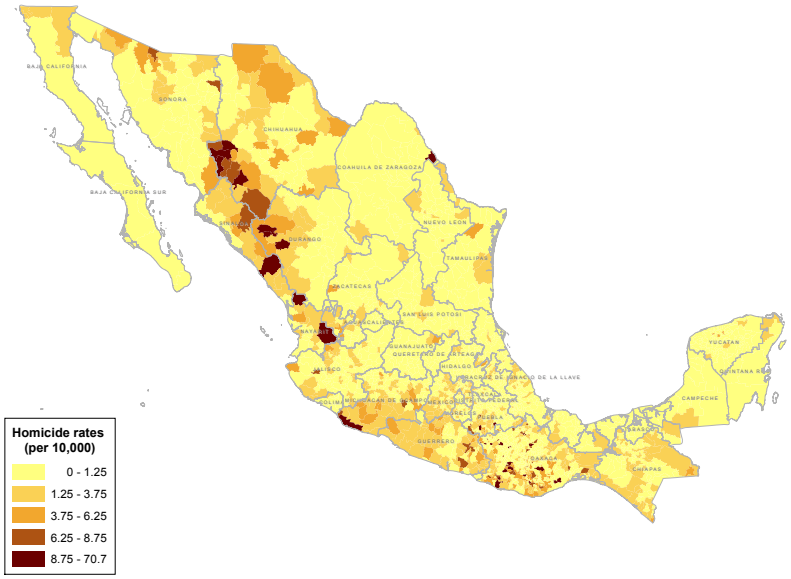


FIGURE 4.3: Municipality Homicide Rates per 10,000 Inhabitants in 2002

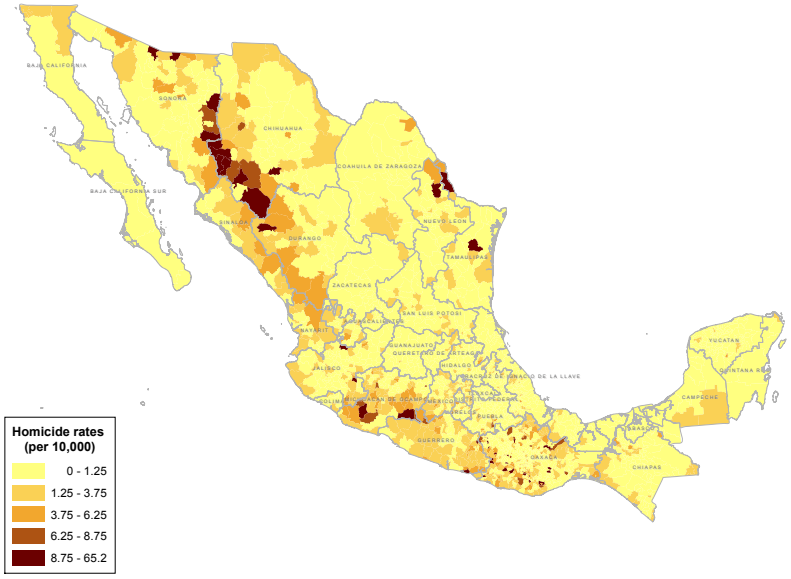


FIGURE 4.4: Municipality Homicide Rates per 10,000 Inhabitants in 2005

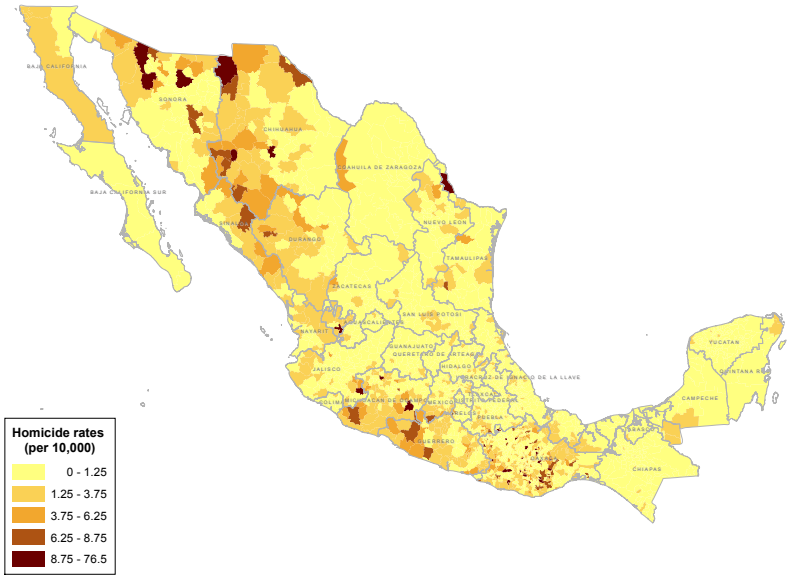


FIGURE 4.5: Municipality Homicide Rates per 10,000 Inhabitants in 2007

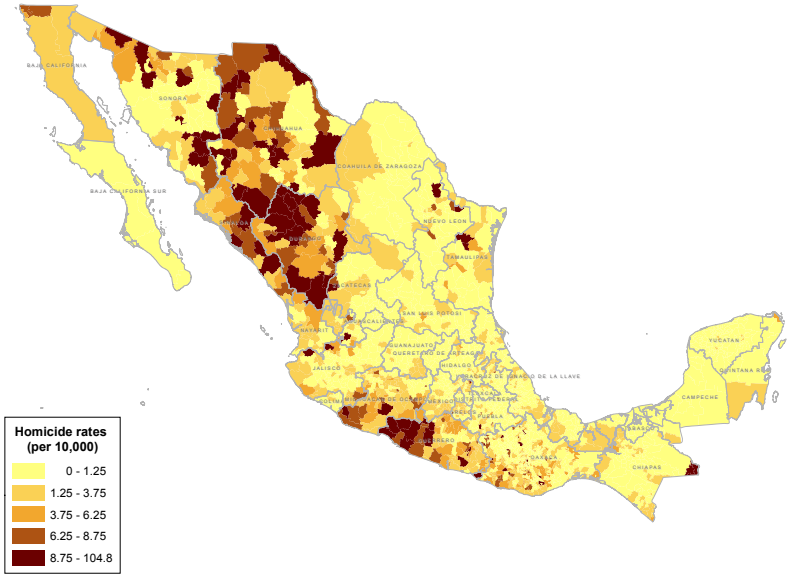


FIGURE 4.6: Municipality Homicide Rates per 10,000 Inhabitants in 2009

Table 4.1: Respondent Attrition and Potential Future Homicide Rate Exposure for Women Aged 7 to 42 in MxFLS1 or Women Aged 10 to 45 in MxFLS2

Homicide Rate	(1)	(2)	Homicide Rate	(3)	(4)
Change Between 2002 & 2009	0.34%		Change Between 2005 & 2009	0.14%	
	(0.21)			(0.16)	
MxFLS1 Characteristics Interacted with Homicide Rate Change Between 2002 & 2009:			*MxFLS2 Characteristics Interacted with Homicide Rate Change Between 2005 & 2009:*		
Age		0.07%	Age		-0.04%
		(0.09)			(0.07)
Age Squared		0.00%	Age Squared		0.00%
		(0.00)			(0.00)
Lived in Rural Locality		0.07%	Lived in Rural Locality		-0.27%
		(0.52)			(0.38)
Year of Education		0.03%	Year of Education		0.00%
		(0.05)			(0.05)
Employed		0.00%	Employed		0.00%
		(0.00)			(0.00)
Earnings per Month (1,000 Pesos)		-0.18%	Earnings per Month (1,000 Pesos)		0.08%
		(0.11)			(0.07)
PCE of HH (10,000 Pesos)		0.37%	PCE of HH (10,000 Pesos)		-0.04%
		(0.42)			(1.07)
Married		0.00%	Married		0.00%
		(0.00)			(0.00)
Household Size		-0.10%	Household Size		0.14%
		(0.11)			(0.08)
Observations	11,511	11,511	Observations	10,429	10,429
Mean of Dependent Variable	13.7%	13.7%	Mean of Dependent Variable	8.4%	8.4%

Notes:

***, ** denote significance at the 1 and 5% level, respectively

Homicide rates are per 10,000. Standard errors are clustered at the municipality level

Regressions include respondent's years of education, marital status, employment status, earnings per month, household per capita expenditure and size, an indicator for whether the municipality is rural, and fixed effects for age, interview year, and month.

Table 4.2: Previous Municipal Trends and Levels of Characteristics' Relationship to Current Homicide Rate

	Municipal Homicide Rate (per 10,000)	
	Level in 2009	Change From 2005 to 2009
Municipality Characteristics	(1)	(2)
CENSUS: Change in Share of Households Between 2000-2005 with:		
Televisions	-6.73	-5.33
	(6.13)	(7.76)
Piped Water	-1.59	4.53
	(5.01)	(5.59)
Sewage System	1.52	-4.62
	(3.74)	(4.44)
Electricity	1.60	7.97
	(9.81)	(11.05)
CENSUS: Change in Share of 21-65 Year Olds Between 2000-2005 with:		
Less Than Primary Education	-0.15	-18.64 *
	(8.27)	(9.97)
At Least High School Diploma	-11.19	-29.55 *
	(13.40)	(15.20)
Speak Indigenous Language	-3.47	-10.25
	(6.91)	(6.76)
Illiterate	-13.38	-22.81
	(27.76)	(31.62)
CENSUS: Change Between 2000-2005 in Share of:		
Less Than 18 Year Olds	7.83	-3.16
	(18.72)	(22.58)
18 to 65 Year Olds	3.79	-10.17
	(27.07)	(29.57)
CENSUS: Change Between 2000-2005 in:		
Average Educational Attainment	1.12	1.02
	(1.46)	(1.61)
MxFLS: Change in Share of Older than 18 Year Olds Between MxFLS1-MxFLS2:		
Married	-4.43	-6.36
	(6.12)	(6.77)
Employed Females	-0.88	1.31
	(4.40)	(4.64)
Employed Males	0.27	1.46
	(4.69)	(4.63)
Self-Employed Females	-2.42	-5.18
	(4.66)	(4.73)
Self-Employed Males	2.89	4.20
	(3.76)	(3.98)
Rural	1.63	2.07 *
	(1.02)	(1.10)
Have Relative in the U.S.	-2.96 *	-2.73
	(1.64)	(1.68)
Have Thoughts of Future Migration	-2.15	-0.12
	(3.34)	(3.55)
Have Fear in the Day	-1.15	0.41
	(6.39)	(6.95)
Have Fear in the Night	-4.70	-6.23
	(6.44)	(6.69)
MxFLS: Change Between MxFLS1-MxFLS2 in:		
Average Household Size	0.03	-0.10
	(0.68)	(0.69)
Log Hourly Earning of Females Older than 18 (Pesos)	0.29	-0.01
	(0.46)	(0.48)
Log Hourly Earning of Males Older than 18 (Pesos)	0.78	0.35
	(0.71)	(0.70)
Log Household Per Capita Expenditure (Pesos)	0.86	1.16
	(1.05)	(1.22)
MxFLS: Change in Share of Localities Between MxFLS1-MxFLS2 with:		
Increased Domestic Violence	-0.05	-0.13
	(0.44)	(0.44)
Presence of Vandalism	0.50	0.35
	(0.38)	(0.43)
Presence of Police	0.19	0.13
	(0.40)	(0.43)
MxFLS: Change Between MxFLS1-MxFLS2 in Localities:		
Number of Primary Schools	0.00	0.00
	(0.00)	(0.00)
Number of Junior Highs	-0.01	0.00
	(0.01)	(0.01)
Number of High Schools	0.01	0.00
	(0.01)	(0.01)
Rate of Poor Households	0.00	-0.01
	(0.01)	(0.01)
Observations	135	135
Mean of Dependent Variable	1.89	0.97
F test: Jointly 0; Prob>F	0.20	0.23

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Robust standard errors provided.

Table 4.3: Descriptive Statistics

	All Births to Panel Members Since MxFLS2		All Births Before 3Q2009 to Panel Members			
			Since MxFLS2		Adds Siblings Born Since Baseline	
Homicide Rate	**Full Sample**	**Sibling Sample**	**Full Sample**	**Sibling Sample**	**Full Sample**	**Sibling Sample**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Birth Outcomes:						
Birth weight in grams (g)	3,241.5	3,205.8	3,249.8	3,238.0	3,241.6	3,249.4
	(619.2)	(600.2)	(624.9)	(595.8)	(609.7)	(585.5)
Low birth weight (<2,500g)	7.7%	7.6%	7.6%	6.2%	7.7%	7.1%
	(26.7)	(26.6)	(26.5)	(24.1)	(26.7)	(25.6)
Low birth weight+ (≤2,500g)	10.6%	12.1%	10.8%	11.1%	10.9%	10.7%
	(30.8)	(32.6)	(31.0)	(31.4)	(31.1)	(30.9)
Premature Birth	19.6%	21.7%	19.8%	19.9%	18.7%	16.5%
	(39.7)	(41.2)	(39.9)	(40.0)	(39.0)	(37.1)
Any Pregnancy Complication	8.1%	5.1%	8.3%	3.6%	8.4%	6.5%
	(27.3)	(22.1)	(27.6)	(18.7)	(27.7)	(24.7)
Male	51.4%	50.7%	50.4%	50.8%	50.3%	50.8%
	(50.0)	(50.0)	(50.0)	(50.1)	(50.0)	(50.0)
Medical Care:						
Born in Hospital	88.9%	85.6%	88.6%	85.0%	88.7%	88.0%
	(31.5)	(35.2)	(31.8)	(35.7)	(31.7)	(32.5)
Number of Prenatal Visits	7.9	7.2	7.9	7.3	7.7	7.3
	(4.1)	(3.3)	(4.0)	(3.4)	(4.0)	(3.7)
Prenatal Care, First 2 Months	66.4%	65.8%	67.5%	68.7%	66.5%	64.3%
	(47.3)	(47.5)	(46.9)	(46.4)	(47.2)	(47.9)
Mother's Characteristics at Baseline:						
Age	19.2	18.0	19.6	18.3	19.6	20.4
	(6.7)	(5.9)	(6.7)	(5.6)	(6.7)	(5.9)
Lived in Rural Locality	51.1%	55.0%	51.1%	55.0%	51.1%	51.6%
	(50.0)	(49.9)	(50.0)	(49.9)	(50.0)	(50.0)
Year of Education	7.5	7.2	7.6	7.4	7.6	7.9
	(3.5)	(3.3)	(3.4)	(3.1)	(3.4)	(3.1)
Employed	31.6%	31.5%	31.0%	29.4%	31.0%	30.0%
	(46.5)	(46.6)	(46.3)	(45.8)	(46.3)	(45.9)
Earnings per Month (Pesos)	575.2	399.3	524.3	380.8	524.3	583.6
	(1,966.5)	(913.1)	(1,899.7)	(897.7)	(1,899.7)	(2,839.3)
PCE of HH (Pesos)	1161.9	946.6	1185.3	974.2	1185.3	1350.2
	(4031.8)	(1176.0)	(4302.2)	(1193.3)	(4302.2)	(5434.6)
Married	28.6%	17.8%	30.0%	19.5%	30.0%	30.2%
	(45.2)	(38.4)	(45.8)	(39.8)	(45.8)	(46.0)
Number of Births	1,850	471	1,548	307	1,868	891
Number of Mothers	1,608	229	1,392	151	1,392	415

Notes:

Standard deviation in parenthesis.

Table 4.4: Respondent Migration Between MxFLS2 and MxFLS3 and Potential Future Homicide Rate Exposure for Panel Women that Gave Birth Since MxFLS2 and before 2Q2009

	Interviewed in Different Municipality		Migrated for at Least 1 Year		Migrated for at Least 1 Month	
Homicide Rate	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Change Between 2005 & 2009	1.18%		1.55% **		1.39% *	
	(0.75)		(0.72)		(0.76)	
MxFLS2 Characteristics Interacted with Homicide Rate Change Between 2005 & 2009:						
Age		-0.03%		0.09%		0.11%
		(0.25)		(0.18)		(0.18)
Age Squared		0.00%		0.00%		0.00%
		(0.00)		(0.00)		(0.00)
Lived in Rural Locality		-0.05%		2.16% **		2.69% ***
		(0.82)		(0.99)		(1.02)
Year of Education		0.05%		-0.05%		-0.07%
		(0.07)		(0.11)		(0.10)
Employed		-0.05%		-0.01%		-0.04%
		(0.10)		(0.23)		(0.21)
Earnings per Month (1,000 Pesos)		0.35%		0.66% *		0.75% *
		(0.23)		(0.37)		(0.44)
PCE of HH (10,000 Pesos)		1.47%		7.02% **		6.62% *
		(2.55)		(2.91)		(3.91)
Married		0.05%		-0.17%		-0.15%
		(0.10)		(0.26)		(0.28)
Household Size		0.03%		-0.23%		-0.16%
		(0.13)		(0.16)		(0.18)
Observations	1,346	1,346	1,277	1,277	1,277	1,277
Mean of Dependent Variable	6.8%	6.8%	15.9%	15.9%	17.4%	17.4%

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000. Standard errors are clustered at the municipality level

Regressions include controls for respondent's MxFLS2 education, age fixed effects, employment status, earnings per month, household size, marital status, household's per capita expenditure, and rural status. Additionally all regressions control for all municipality characteristic trends and levels shown in Table 2, as well as, year of interview, month of interview, and MxFLS2 state of residence fixed effects.

Table 4.5: Impact of Local Homicide Rate on Birth Rate at the Municipality-Month Level from January 2003 to June 2009

	Birth Rate of 14-49 Yr. Old Women (per 1,000)		
	All	<9 Yrs. Education	≥9 Yrs. Education
Homicide Rate	(1)	(2)	(3)
15 Months Before Birth	-0.23	0.08	-0.55 **
	(0.19)	(0.37)	(0.25)
14 Months Before Birth	0.11	0.80	-0.50
	(0.27)	(0.52)	(0.25)
13 Months Before Birth	0.15	0.68	-0.31
	(0.26)	(0.48)	(0.24)
12 Months Before Birth	-0.14	0.17	-0.57 **
	(0.26)	(0.38)	(0.24)
11 Months Before Birth	-0.17	0.40	-0.17
	(0.20)	(0.43)	(0.26)
10 Months Before Birth	-0.10	-0.10	-0.22
	(0.28)	(0.44)	(0.24)
9 to 7 Months Before Birth	0.00	0.20	0.19
	(0.14)	(0.22)	(0.25)
6 to 4 Months Before Birth	-0.19	0.04	-0.36 **
	(0.16)	(0.25)	(0.17)
3 to 1 Months Before Birth	0.12	0.38	0.11
	(0.18)	(0.38)	(0.27)
Mean Monthly Birth Rate (1,000)	2.7	3.8	2.7
Municipality-Months	10,452	10,452	10,451

Notes:

***, ** denote significance at the 1 and 5% level, respectively. Homicide rates are per 10,000.

Robust standard errors provided. Regressions include year, month, municipality, and state interacted with year fixed effects.

Table 4.6: Impact of Local Homicide Rate on Birth Weight
for All Births to Panel Members Since MxFLS2 and Before July 2009, Adding Siblings Born Since MxFLS1

Homicide Rate	(1)	(2)	(3)	(4)	(5)	(6)	(7)
9 to 7 Months Before Birth	-0.9	7.3	-46.9	-120.3 **	-222.5 **	-226.9 **	-299.9 ***
	(34.4)	(56.8)	(75.1)	(60.3)	(97.4)	(91.5)	(106.0)
6 to 4 Months Before Birth	-61.5 **	-66.0	-89.4	31.7	53.9	38.9	118.3
	(29.7)	(40.3)	(78.2)	(58.8)	(71.4)	(69.7)	(84.8)
3 to 1 Months Before Birth	35.6	-2.8	15.9	21.8	121.4	67.7	6.2
	(35.1)	(48.6)	(75.6)	(59.2)	(103.1)	(79.0)	(86.1)
Sibling Sample	NO	NO	YES	YES	YES	YES	YES
Sibling Fixed Effects	NO	NO	NO	YES	YES	YES	YES
Municipality Fixed Effects	NO	YES	YES	NO	YES	YES	YES
Pre/Post Gestation Hom Rates	NO	YES	YES	NO	NO	YES	YES
State and YOB Fixed Effects	NO	YES	YES	NO	NO	NO	YES
Mean of Dependent Variable	3241.6	3241.6	3,249.4	3,249.4	3,249.4	3,249.4	3,249.4
Observations	1,868	1,868	891	891	891	891	891
Number of Mothers				415	415	415	415

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000.

Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.7: Impact of Local Homicide Rate on Birth Weight
for All Births to Panel Members Since MxFLS2, Adding Siblings Born Since MxFLS1

	All Births to Panel Members Since MxFLS2 and Before3Q2009			
	All	Before 2Q2009	Before 2009	In Clinic or Hospital
Homicide Rate	**(1)**	**(2)**	**(3)**	**(4)**
9 to 7 Months Before Birth	-299.9 ***	-282.0 **	-324.0 **	-348.4 *
	(106.0)	(122.5)	(133.5)	(197.6)
6 to 4 Months Before Birth	118.3	32.8	44.4	91.3
	(84.8)	(91.2)	(97.0)	(152.5)
3 to 1 Months Before Birth	6.2	34.9	-55.0	93.0
	(86.1)	(125.1)	(121.5)	(159.2)
Sibling Sample	YES	YES	YES	YES
Sibling Fixed Effects	YES	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES	YES
Pre/Post Gestation Hom Rates	YES	YES	YES	YES
State and YOB Fixed Effects	YES	YES	YES	YES
Mean of Dependent Variable	3,249.4	3,257.0	3,248.6	3,251.3
Observations	891	809	735	727
Number of Mothers	415	377	343	339

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000.

Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.8: Impact of Local Homicide Rate on Birth Outcomes
for All Births to Panel Members Since MxFLS2 and Before July 2009, Adding Siblings Born Since MxFLS1

Homicide Rate	LBW (<2,500g) (1)	LBW+ (≤2,500g) (2)	Child Premature (3)	Complications (4)
9 to 7 Months Before Birth	10.7% **	21.2% ***	4.2%	1.7%
	(5.0)	(7.7)	(6.7)	(5.8)
6 to 4 Months Before Birth	-4.1%	-5.1%	-9.5% **	-3.5%
	(3.7)	(5.4)	(4.4)	(3.7)
3 to 1 Months Before Birth	-0.3%	3.4%	11.4% *	0.5%
	(4.2)	(7.4)	(6.5)	(5.5)
Sibling Sample	YES	YES	YES	YES
Sibling Fixed Effects	YES	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES	YES
Pre/Post Gestation Hom Rates	YES	YES	YES	YES
State and YOB Fixed Effects	YES	YES	YES	YES
Mean of Dependent Variable	7.1%	10.7%	16.5%	6.5%
Observations	891	891	891	886
Number of Mothers	415	415	415	413

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000.

Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.9: Impact of Local Homicide Rate on Birth Weight
for Births to Subgroups of Panel Members Since MxFLS2 and Before July 2009, Adding Siblings Born Since MxFLS1

	All	Bottom 50% PCE in MxFLS1	<9 Yrs. Education in MxFLS1	<9 Yrs. Education in MxFLS3
Homicide Rate	**(1)**	**(2)**	**(3)**	**(4)**
9 to 7 Months Before Birth	-226.9 **	-501.9 **	-500.5 ***	-475.1 ***
	(91.5)	(240.1)	(129.7)	(162.2)
6 to 4 Months Before Birth	38.9	38.2	38.6	188.7
	(69.7)	(126.5)	(103.6)	(147.1)
3 to 1 Months Before Birth	67.7	-60.7	-45.4	-85.6
	(79.0)	(157.1)	(126.4)	(173.4)
Sibling Sample	YES	YES	YES	YES
Sibling Fixed Effects	YES	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES	YES
Pre/Post Gestation Hom Rates	YES	YES	YES	YES
State and YOB Fixed Effects	NO	NO	NO	NO
Mean of Dependent Variable	3,249.4	3,231.1	3,214.5	3,221.6
Observations	891	432	487	364
Number of Mothers	415	203	224	165

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000. Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.10: Impact of Local Homicide Rate on Prenatal Care Visits
for All Births to Panel Members Since MxFLS2 and Before July 2009, Adding Siblings Born Since MxFLS1

	All	Bottom 50% PCE in MxFLS1	<9 Yrs. Education in MxFLS1	<9 Yrs. Education in MxFLS3
Homicide Rate	**(1)**	**(2)**	**(3)**	**(4)**
9 to 7 Months Before Birth	-1.2 **	-2.7 ***	-1.7	-2.7 **
	(0.6)	(0.8)	(1.1)	(1.1)
6 to 4 Months Before Birth	-0.3	-0.5	0.2	0.4
	(0.5)	(0.6)	(0.4)	(0.7)
3 to 1 Months Before Birth	-0.1	2.2 *	2.1 **	2.8 ***
	(0.5)	(1.2)	(1.0)	(1.0)
Sibling Sample	YES	YES	YES	YES
Sibling Fixed Effects	YES	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES	YES
Pre/Post Gestation Hom Rates	YES	YES	YES	YES
State and YOB Fixed Effects	YES	NO	NO	NO
Mean of Dependent Variable	7.3	6.9	7.0	6.9
Observations	886	429	482	361
Number of Mothers	413	202	222	164

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000. Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.11: Impact of Local Homicide Rate on Prenatal Care Initiation in the First Two Months of Pregnancy for All Births to Panel Members Since MxFLS2 and Before July 2009, Adding Siblings Born Since MxFLS1

Homicide Rate	All (1)	Bottom 50% PCE in MxFLS1 (2)	<9 Yrs. Education in MxFLS1 (3)	<9 Yrs. Education in MxFLS3 (4)
9 to 7 Months Before Birth	-29.4% ***	-36.0% ***	-15.8%	-9.6%
	(9.8)	(11.2)	(16.5)	(19.8)
6 to 4 Months Before Birth	13.7%	-10.6%	10.3%	-8.4%
	(8.7)	(10.4)	(9.2)	(14.0)
3 to 1 Months Before Birth	3.9%	38.3% **	16.3%	38.2% **
	(9.2)	(16.1)	(13.1)	(17.1)
Sibling Sample	YES	YES	YES	YES
Sibling Fixed Effects	YES	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES	YES
Pre/Post Gestation Hom Rates	YES	YES	YES	YES
State and YOB Fixed Effects	YES	NO	NO	NO
Mean of Dependent Variable	64.3%	63.1%	60.9%	60.1%
Observations	889	432	485	362
Number of Mothers	414	203	223	164

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000. Standard errors are clustered at the municipality level. Regressions additionally include controls for the gender of the child, maternal (age at birth, age at birth squared, years of education, employment status, earnings per month, and marital status) and household characteristics (size, per capita expenditure, and rural status), as well as, year of birth, month of birth, year of interview, month of interview, birth order, and survey wave fixed effects.

Table 4.12: Impact of Local Homicide Rate on Maternal Health Behaviors for Mothers of the Sibling Sample used in Table 4.6

Homicide Rate	Number of Cigarettes Smoked Per Week (1)	Number of Exercise Days Between Monday and Friday (2)	Amount of Exercise Time Per Day (Mins) (3)
1- 12 Months Before Interview	-0.15	0.06 *	1.35
	(0.14)	(0.04)	(1.41)
Individual Fixed Effects	YES	YES	YES
Municipality Fixed Effects	YES	YES	YES
Mean of Dependent Variable	0.5	0.35	9.21
Observations	768	768	768
Number of Mothers	384	384	384

Notes:

***, **, * denote significance at the 1, 5, and 10% level, respectively. Homicide rates are per 10,000.

Standard errors are clustered at the municipality level. Regressions additionally include controls for time-varying individual (age, years of education, marital status, employment status, earnings per month) and household characteristics (size, per capita expenditure, rural status), as well as, year of interview and month of interview fixed effects.

5

Conclusion

The research in this dissertation makes two major contributions to the study of the impact of early-life influences on later life wellbeing. The first contribution is the reevaluation of the seminal work in this field which connects the in utero environment to adult economic outcomes. While this esteemed work represented a large step forward in the econometric rigor needed to identify how a change in the disease environment a mother faces may impact the livelihood of the in utero child, its failure to control for the large demographic changes in its sample population documented in this dissertation, call its results into question.

While this analysis in no way comments on the overall legitimacy of the fetal-origins hypothesis, it does assert that its most influential work linking early-life health to adult economic outcomes has large enough identification ambiguity to make its estimates untenable. Specifically, the findings in this analysis, indicate that those exposed in utero to the 1918 influenza pandemic have equivalent later life socioeconomic status as surrounding cohorts. Interestingly, this result may provide supportive evidence that there is scope for post-birth intervention to mitigate the adverse impacts of early life health insults on long-term economic well-being.

The second contribution of this dissertation is to explore, using the most thorough non-experimental techniques to date in this literature, whether a mother's psychological wellbeing during pregnancy has an impact on the in utero child's early-life health. Using unanticipated events in both a developed and developing country setting, this dissertation finds that there is a statistically significant relationship between maternal mental health and the birth outcomes of the in utero child. Moreover, this research provides evidence that when the psychological distress is large enough, as in the case of Mexican women at risk of victimization from the Mexican drug cartels, the effect is also economically significant.

Specifically, the analysis in this dissertation finds that the average increase in Mexico to exposure to local violence in early gestation leads to a 75 gram reduction in birth weight and a 40-50% increased risk of being born low birth weight. The large magnitude of the effect on birth weight is further exacerbated amongst those of lower socioeconomic status, with the children of this group of exposed mothers facing decreases of around 120-125 grams on average. These effect size are on the magnitude but in opposite direction of the benefits of a mother being enrolled in large federal health and nutrition programs, suggesting maternal mental health is an important factor in the early-life wellbeing of the in utero child.

Bibliography

Almond, D. (2006), "Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population," *Journal of Political Economy*, 114, 672–712.

Almond, D. and Currie, J. (2011), "Killing Me Softly: The Fetal Origins Hypothesis," *Journal of Economic Perspectives*, 25, 153–72.

Almond, D. and Mazumder, B. (2005), "The 1918 Influenza Pandemic and Subsequent Health Outcomes: An Analysis of SIPP Data," *American Economic Review Papers and Proceedings*, 95, 258–62.

Almond, D. and Mazumder, B. (2011), "Health capital and the prenatal environment: the effect of Ramadan observance during pregnancy," *American Economic Journal: Applied Economic*, 3, 56–85.

Almond, D., Chay, K. Y., and Lee, D. S. (2005), "The costs of low birth weight," *The Quarterly Journal of Economics*, 120, 1031–1083.

Almond, D., Hoynes, H., and Schanzenbach, D. (2011), "Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes," *The Review of Economics and Statistics*, 93, 387–403.

Barber, S. and Gertler, P. (2008), "The Impact of Mexico's Conditional Cash Transfer Programme, Oportunidades, on Birthweight," *Tropical Medicine and International Health*, 13, 1405–1414.

Barker, D. (1994), *Mothers, Babies, and Disease in Later Life*, BMJ Publishing Group, London.

Becker, G. (1960), "An Economic Analysis of Fertility," in *Demographic and Economic Change in Developed Countries*, Princeton University Press, Princeton.

Becker, G. and Rubinstein, Y. (2011), "Fear and Response to Terrorism: An Economic Analysis," Brown University Working Paper.

Behrman, J. and Rosenzweig, M. (2004), "Returns to Birthweight," *The Review of Economics and Statistics*, 86, 586–601.

Berkowitz, G., Wolff, M., Janevic, T., Holzman, I., Yehuda, R., and Landrigan, P. (2003), "The World Trade Center disaster and intrauterine growth restriction," *JAMA*, 290, 595–596.

Beydoun, H. and Saftlas, A. (2008), "Physical and Mental Health Outcomes of Prenatal Maternal Stress in Humans and Animal Studies: A Review of Recent Evidence," *Paediatric and Perinatal Epidemiology*, 22, 438–466.

Bharadwaj, P., Eberhard, J., and Nelson, C. (2010), "Do Initial Endowments Matter Only Initially? Birth Weight, Parental Investments and Academic Achievement in School," working paper, University of California-San Diego.

Black, S., Devereux, P., and Salvanes, K. (2007), "From the Cradle to the Market? The Effect of Birth Weight on Adult Outcomes," *Quarterly Journal of Economics*, 122, 409–39.

Brainerd, E. and Siegler, M. (2003), "The Economic Effects of the 1918 Influenza Epidemic," *Discussion Paper no. 3791, Centre Econ. Policy Res*, 13.

Bram, J., Orr, J., and Rapaport, C. (2002), "Measuring the Effect of the September 11 Attack on New York City," *Federal Reserve Bank of New York Economic Policy Review*, 8, 5–20.

Brooke, O., Anderson, H., Bland, J., Peacock, J., and Stewart, C. (1989), "Effects on Birth Weight of Smoking, Alcohol, Caffeine, Socioeconomic Factors, and Psychological Stress," *British Medical Journal*, 298, 795–801.

Brooks-Gunn, J. and Duncan, G. (1997), "The Effects of Poverty on Children," *The Future of Children*, 7, 55–71.

Brown, R. (2014), "The Intergenerational Impact of Terror: The Extended Reach of the 9/11 Tragedy," Duke University Working Paper.

Brown, R. and Velasquez, A. (2014), "The Effect of Violent Conflict in Mexico on the Human Capital Accumulation of Children and Young Adults," Duke University Working Paper.

Buckles, K. S. and Hungerman, D. M. (2013), "Season of birth and later outcomes: Old questions, new answers," *Review of Economics and Statistics*, 95, 711–724.

Camacho, A. (2008), "Stress and Birth Weight: Evidence from Terrorist Attacks," *American Economic Review: Papers & Proceedings*, 98, 511–515.

Case, A., Fertig, A., and Paxson, C. (2005), "The Lasting Impact of Childhood Health and Circumstance," *Journal of Health Economics*, 24, 365–389.

Castillo, J., Mejia, D., and Restrepo, P. (2013), "Illegal Drug Markets and Violence in Mexico: The Causes Beyond Caldern," Mimeo, Universidad de los Andes.

Catalano, R., Bruckner, T., Marks, A., and Eskenazi, B. (2006), "Exogenous Shocks to the Human Sex Ratio: the Case of September 11, 2001 in New York City," *Human Reproduction*, 21, 3127–3131.

Corcoran, M., Gordon, R., Laren, D., and Solon, G. (1992), "The Association Between Men's Economic Status and Their Family and Community Origins," *J. Hum. Resour*, 27, 575–601.

Crawford, R. (1995), "The Spanish Flu," in *Stranger Than Fiction: Vignettes of San Diego History*, San Diego Historical Society, San Diego.

Davis-Kean, P. (2005), "The Influence of Parent Education and Family Income on Child Achievement: The Indirect Role of Parental Expectations and the Home Environment," *Journal of Family Psychology*, 19, 294–304.

de Catanzaro D. and Macniven, E. (1992), "Psychogenic pregnancy disruptions in mammals," *Neuroscience & Biobehavioral Reviews*, 16, 43–53.

de Weerth C. and Buitelaar, J. (2005), "Physiological stress reactivity in human pregnancy–a review," *Neuroscience & Biobehavioral Reviews*, 29, 295–312.

Deimling, G. and Bass, D. (1986), "Symptoms of Mental Impairment Among Elderly Adults and their Effects on Family Caregivers," *Journal of Gerontology: Social Sciences*, 41, 778–785.

Dell, M. (2011), "Trafficking Networks and the Mexican Drug War," Mimeo. Harvard University.

Diaz-Cayeros, A., Magaloni, B., Matanock, A., and Romero, V. (2011), "Living in Fear: Mapping the Social Embeddedness of Drug Gangs and Violence in Mexico," mimeo.

Dole, N., Savitz, D., Siega-Riz, A., McMahon, M., and Buckens, P. (2003), "Maternal Stress and Preterm Birth," *American Journal of Epidemiology*, 157, 14–24.

Dolfman, M. and Wasser, S. (2004), "9/11 and the New York City Economy," *Monthly Labor Review*, 127, 3–33.

Duflo, E. (2000), "Child Health and Household Resources in South Africa: Evidence from the Old Age Pension Program," *American Economic Review Papers and Proceedings*, 90, 393–98.

Eccleston, M. (2011), *In Utero Exposure to Maternal Stress: Effects of the September 11th terrorist attacks in New York City on Birth and Early Schooling Outcomes*, Harvard University. Retrieved from mecclest/papers/jmp.pdf, Cambridge, MA.

El-Sayed, A., Hadley, C., and Galea, S. (2008), "Birth Outcomes Among Arab Americans in Michigan Before and After the Terrorist Attacks of September 11, 2001," *Ethnicity & Disease*, 18, 348–56.

Endara, S., Ryan, M., Sevick, C., Conlin, A., and Macera, C. (2009), "Does Acute Maternal Stress in Pregnancy Affect Infant Health Outcomes? Examination of a Large Cohort of Infants Born After the Terrorist Attacks of September 11, 2001," *BMC Public Health*, 9.

Eskenazi, B., Marks, A., Catalano, R., Bruckner, T., and Toniolo, P. (2007), "Low Birthweight in New York City and Upstate New York Following the Events of September 11th," *Human Reproduction*, 22, 3013–3020.

Evans, R. W., Hu, Y., and Zhao, Z. (2010), "'The Fertility Effect of Catastrophe: U.S. Hurricane Births," *Journal of Population Economics*, 23, 1–36.

Figlio, D., Guryan, J., Krzysztof, K., and Roth, J. (2013), "The Effects of Poor Neonatal Health on Children's Cognitive Development," Working Paper 18846, NBER.

Glynn, L., Wadhwa, P., Dunkel-Schetter, C., Chicz-DeMet, A., and Sandman, C. (2001), "When Stress Happens Matters: Effects of Earthquake Timing on Stress Responsivity in Pregnancy," *American Journal of Obstetrics and Gynecology*, 184, 637–42.

Guerrero-Gutiérrez, E. (2011), "Security, Drugs, and Violence in Mexico: A Survey," in *7th North American Forum, Washington DC*.

Hedegaard, M., Henriksen, T., Sabroe, S., and Secher, N. (1993), "Psychological Distress in Pregnancy and Preterm Delivery," *BMJ*, 307, 234–39.

Hedegaard, M., Henriksen, T., and Sabroe, S. (1996), "The Relationship Between Psychological Distress During Pregnancy and Birth Weight for Gestational Age," *Acta Obstetricia et Gynecologica Scandinavica*, 75, 32–39.

Hill, M. and Duncan, G. (1987), "Parental Family Income and the Socioeconomic Attainment of Children," *Soc. Sci. Res*, 16, 37–73.

Hoynes, H., Page, M., and Stevens, A. (2011), "Can Targeted Transfers Improve Birth Outcomes?: Evidence from the Introduction of the WIC Program," *Journal of Public Economics*, 95, 813–827.

Instituto Nacional de Estadistica Geografia e Informatica. (2003), *Sintesis Metodologica de las Estadisticas Vitales.*, Instituto Nacional de Estadistica Geografia e Informatica., Mexico: INEGI.

Jewell, R. (2007), "Prenatal Care and Birthweight Production: Evidence from South America," *Applied Economics*, 39, 415–426.

Jewell, R. and Triunfo, P. (2006), "The Impact of Prenatal Care on Birthweight: the case of Uruguay," *Health Economics*, 15, 1245–1250.

Kannisto, V., Christensen, K., and Vaupel, J. (1997), "No Increased Mortality in Later Life for Cohorts Born during Famine," *American J. Epidemiology*, 145, 987–94.

Kaushal, N., Kaestner, R., and Reimers, C. (2007), "Labor market effects of September 11th on Arab and Muslim residents of the United States," *Journal of Human Resources*, 42, 275–308.

Knudsen, H. K., Roman, P. M., Johnson, J. A., and Ducharme, L. J. (2005), "A Changed America? The Effects of September 11th on Depressive Symptoms and Alcohol Consumption," *Journal of Health and Social Behavior*, 46, 260–273.

Landrigan, P. (2001), "Health Consequences of the 11 September 2001 Attacks," *Environmental Health Perspectives*, 109, A514–15.

Landrigan, P., Lioy, P., Thurston, G., Berkowitz, G., Chen, L., Chillrud, S., Gavett, S., Georgopoulos, P., Geyh, P., Levin, S., Perera, F., Rappaport, S., Small, C., and the NIEHS World Trade Center Working Group (2004), "Health and Environmental Consequences of the World Trade Center Disaster," *Environmental Health Perspectives*, 112, 731–39.

Lauderdale, D. (2006), "Birth Outcomes for Arabic-Named Women in California Before and After September 11th," *Demography*, 43, 185–201.

Lederman, S., Rauh, V., Weiss, L., Stein, J., Hoepner, L., Becker, M., and Perera, F. (2004), "The Effects of the World Trade Center Event on the Birth Outcomes Among Term Deliveries at the Three Lower Manhattan Hospitals," *Environmental Health Perspectives*, 112, 1772–1778.

Mancuso, R., Schetter, C., Rini, C., Roesch, S., and Hobel, C. (2004), "Maternal Prenatal Anxiety and Corticotropin-Releasing Hormone Associated with Timing of Delivery," *Psychosom Med*, 66, 762–69.

Mansour, H. and Rees, D. (2012), "Armed conflict and birth weight: Evidence from the al-Aqsa Intifada," *Journal of Development Economics*, 99, 190–199.

Martin, J., Hamilton, B., Ventura, S., Menacker, F., Park, M., and Sutton, P. (2002), "Births: Final Data for 2001," *National Vital Statistics Report*, 51, 1–104.

McEwen, B. S. (1998), "Protective and Damaging Effects of Stress Mediators," *The New England Journal of Medicine*, 338, 171–179.

Molzahn, C., Rios, V., and Shirk, D. (2012), *Drug Violence in Mexico: Data and Analysis Through 2011*, Trans Border Institute Joan B. Kroc School of Peace Studies University of, San Diego.

Mulder, E., Robles de Medina, P., Huizink, A., Van den Bergh, B., Buitelaar, J., and Visser, G. (2002), "Prenatal Maternal Stress: Effects on Pregnancy and the (Unborn) Child," *Early Human Development*, 70, 3–14.

Neugebauer, R., Hoek, H., and Susser, E. (1999), "Prenatal Exposure to Wartime Famine and Development of Antisocial Personality Disorder in Early Adulthood," *J. American Medical Assoc*, 282, 455–62.

Newton, R. and Hunt, L. (1984), "Psychosocial Stress in Pregnancy and its Relation to Low Birth Weight," *BMJ (Clin Res Ed)*, 288, 1191–1194.

Noelker, L. and Townsend, A. (1987), "Perceived Caregiving Effectiveness: The Impact of Parental Impairment, Community Resources, and Caregiver Characteristics," in *Aging, Health, and Family: Long-Term Care*, ed. T. H. Brubaker, pp. 58–79, Sage, Newbury Park, CA.

Nudd, J. (2004), "U.S. World War I Draft Registrations," *Yesterdays*, 24, 34–41.

Park, M. and Falco, M. (2010), "Older Mothers' Kids Have Higher Autism Risk, Study Finds," http://www.cnn.com/.

Perera, F., Tang, D., Rauh, V., Lester, K., Tsai, W., Tu, Y., Weiss, L., Hoepner, L., King, J., Del Priore, G., and Lederman, S. (2005), "Relationships Among Polycyclic Aromatic Hydrocarbon-DNA Adducts, Proximity to the World Trade Center and Effects on Fetal Growth," *Environmental Health Perspectives*, 113, 1062–1067.

Rasmussen, K. (2001), "The 'Fetal Origins' Hypothesis: Challenges and Opportunities for Maternal and Child Nutrition," *Ann. Rev. Nutrition*, 21, 73–95.

Ravelli, A. C., van der Meulen, J. H., Osmond, C., Barker, D. J., and Bleker, O. P. (1999), "Obesity at the age of 50 y in men and women exposed to famine prenatally," *The American journal of clinical nutrition*, 70, 811–816.

Reichman, N. and Hade, E. (2001), "Validation of Birth Certificate Data. A Study of Women in New Jersey's HealthStart Program," *Annals of Epidemiology*, 11, 186–93.

Rich-Edwards, J., Kleinman, K., Strong, E., Oken, E., and Gillman, M. (2005), "Preterm Delivery in Boston Before and After September 11th, 2001," *Epidemiology*, 16, 323–27.

Rios, V. and Shirk, D. (2012), *Drug Violence in Mexico: Data and Analysis through 2010*, Trans-Border Insitute Joan B. Kroc School of Peace Studies, University of San Diego.

Robles, G., Calderon, G., and Magaloni, B. (2013), "The Economic Consequences of Violence and Crime in Mexico," Working paper for the costs of crime and violence in latin american and the caribbean: Methodological innovations and new dimensions, IDB.

Roseboom, T. J., van der Meulen, J. H., Ravelli, A. C., van Montfrans, G. A., Osmond, C., Barker, D. J., and Bleker, O. P. (1999), "Blood pressure in adults after prenatal exposure to famine," *Journal of hypertension*, 17, 325–330.

Roseboom, T. J., van der Meulen, J. H., Osmond, C., Barker, D. J., Ravelli, A. C., Schroeder-Tanka, J. M., van Montfrans, G. A., Michels, R. P., and Bleker, O. P. (2000), "Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45," *Heart*, 84, 595–598.

Rosenzweig, M. and Schultz, T. (1983), "Estimating a Household Production Function: Heterogeneity, the Demand for Health Inputs, and Their Effects on Birth Weight," *Journal of Political Economy*, 91, 723–746.

Rosenzweig, M. and Zhang, J. (2012), "Economic Growth, Comparative Advantage, and Gender Difference in Schooling Outcomes: Evidence from the Birthweight Differences of Chinese Twins," Working paper, Yale University Economics Department.

Schneider, M., Roughton, E., Koehler, A., and Lubach, G. (1999), "Growth and Development Following Prenatal Stress Exposure in Primates: An Examination of Ontogenetic Vulnerability," *Child Development*, 70, 263–74.

Schulte, H., Weisner, D., and Allolio, B. (1990), "The Corticotropin Releasing Hormone Test in Late Pregnancy: lack of Adrenocorticotropin and Cortisol Response," *Clinical Endocrinology*, 33, 99–106.

Schuster, M., Stein, B., Jaycox, L., Collins, R., Marshall, G., Elliott, M., Zhou, A., Kanouse, D., Morrison, J., and Berry, S. (2001), "A National Survey of Stress Reactions After the September 11, Terrorist Attacks," *New England Journal of Medicine*, 345, 1507–1512.

Schwarz, G. (1978), "Estimating the Dimension of a Model," *The Annals of Statistics*, 6, 461–464.

Sexton, M. and Hebel, J. (1984), "A Clinical Trial of Change in Maternal Smoking and its Effect on Birth Weight," *The Journal of the American Medical Association*, 251, 911–915.

Silver, R., Holman, E., McIntosh, D., Poulin, M., and Gil-Rivas, V. (2002), "Nationwide Longitudinal Study of Psychological Responses to September 11," *JAMA*, 288, 1235–1244.

Smits, L., Krabbendam, L., de Bie, R., Essed, G., and van Os, J. (2006), "Lower Birth Weight of Dutch Neonates who were in Utero at the Time of the 9/11 Attacks," *Journal of Psychosomatic Research*, 61, 715–17.

Stanner, S. A., Bulmer, K., Andres, C., Lantseva, O. E., Borodina, V., Poteen, V., and Yudkin, J. S. (1997), "Does malnutrition in utero determine diabetes and coronary heart disease in adulthood? Results from the Leningrad siege study, a cross sectional study," *BMJ: British Medical Journal*, pp. 1342–1348.

Stein, B., Elliott, M., Jaycox, L., Collins, R., Berry, S., Klein, D., and Schuster, M. (2004), "A National Survey of Psychological Consequences of the September 11, 2001 Terrorist Attacks: Reactions, Impairment, and Help-Seeking," *Psychiatry*, 67, 105–117.

Stoller, E. and Pugliesi, K. (1989), "Other Roles of Caregivers: Competing Responsibilities or Supportive Resources," *Journal of Gerontology: SocialSciences*, 44, 231–238.

Strauss, J. and Thomas, D. (2007), "Chapter 54: Health Over the Life Course," *Handbook of Development Economics*, 4, 3375–3474.

Thomas, D. (2010), "Health and Socioeconomic Status: The Importance of Causal Pathways," in *World Bank Annual Conference on Development Economics*, eds. B. Pelskovic and J. Y. Lin.

Thomas, D. and Strauss, J. (1998), "Health, Nutrition, and Economic Development," *Journal of Economic Literature*, 36, 766–817.

Torche, F. (2011), "The Effect of Maternal Stress on Birth Outcomes: Exploiting a Natural Experiment," *Demography*, 48, 1471–1493.

Torche, F. and Echevarria, G. (2011), "The Effect of Birthweight on Childhood Cognitive Development in a Middle-Income Country," *International Journal of Epidemiology*, 40, 1008–1018.

Torche, F. and Kleinhaus, K. (2012), "Prenatal Stress, Gestational Age and Secondary Sex Ratio: The Sex-Specific Effects of Exposure to a Natural Disaster in Early Pregnancy," *Human Reproduction*, 27, 558–67.

Torche, F. and Villarreal, A. (2012), "Prenatal exposure to violence and birth weight in Mexico: Selectivity, exposure, and behavioral responses," Working Paper.

Velasquez, A. (2013), "The Economic Burden of Crime," Duke University Working Paper.

Wadhwa, P., Sandman, C., Porto, M., Dunkel-Schetter, C., and Garite, T. (1993), "The Association Between Prenatal Stress and Infant Birth Weight and Gestational Age at Birth: A Prospective Investigation," *American Journal of Obstetrics and Gynecology*, 169, 858–65.

Wadhwa, P., Culhane, J., Rauh, V., and Barve, S. (2001), "Stress and Preterm Birth: Neuroendocrine, Immune/Inflammatory, and Vascular Mechanisms," *Maternal and Child Health Journal*, 5, 119–25.

Wadhwa, P., Garite, T., Porto, M., Glynn, L., Chicz-DeMet, A., Dunkel-Schetter, C., and Sandman, C. (2004), "Placental Corticotropin-Releasing Hormone (CRH), Spontaneous Preterm Birth, and Fetal Growth Restriction: A Prospective Investigation," *American Journal of Obstetrics and Gynecology*, 191, 1059–60.

Ward, C., Lewis, S., and Coleman, T. (2007), "Prevalence of Maternal Smoking and Environmental Tobacco Smoke Exposure During Pregnancy and Impact on Birth Weight:Retrospective Study Using Millennium Cohort," *BMC Public Health*, 7, 1–7.

Wehby, G., Murray, J., Castilla, E., Lopez-Camelo, J., and Ohsfeldt, R. (2009), "Quatile effects of prenatal care utilization on birth weight in Argentina," *Health Economics*, 18, 1307–1321.

Wilcox, A. (1993), "Birth Weight and Perinatal Mortality: The Effect of Maternal Smoking," *American Journal of Epidemiology*, 137, 1098–1104.

Biography

Ryan Palmer Brown, born April 26th, 1982 in Allentown, PA, received a Ph.D. and M.A. in Economics from Duke University and a B.A. in Economics and Psychology from Vassar College. He is an applied microeconomist with research interests in the fields of health, development, and labor economics, whose work focuses on disentangling and mechanizing the role of various early life and adolescent inputs and environments on later life health, cognition, and wealth. His current research agenda applies econometric techniques to population representative data in both developed and developing country settings in order to rigorously examine the impact of the social, physical, and psychological environment on the human capital accumulation and long-term economic outcomes of children and young adults. His work has been supported by a T32 training grant in the Social, Medical, and Economic Demography of Aging from the National Institute on Aging/National Institutes of Health and has been honored with the 2012 IPUMS Research Award and the NSF/NBER Crime Research Fellowship. Starting in the fall of 2014 he will be an Assistant Professor of Economics at the University of Colorado, Denver.